Dissertation on

"Predictor of HbA1C in abnormal OGCT in 1st and 2nd trimester and its correlation with abnormal fetal outcome in comparison with ultra sonogram"

Submitted to the

The Tamil Nadu Dr. M.G.R. Medical University

In partial fulfilment of the requirements
For the award of degree of

MD [BRANCH II]

OBSTETRICS & GYNECOLOGY

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
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APRIL 2012

ACKNOWLEDGEMENT

I extend my gratitude to the ***Dean Prof Dr. V. KANAGASABAI M.D.*** Madras Medical college, Chennai, for his kind permission to do this dissertation and to use the hospital resource for this study.

I am extremely thankful and grateful to my respected ***Director Prof. Dr. P. M. GOPINATH M.D., D.G.O.*** Institute of Social Obstetrics and Government Kasturba Gandhi Hospital, Chennai for providing with the necessary facilities to carry out this study and for her continuous support and guidance.

I express my gratitude and thanks to ***Director Prof. Dr. M. MOHAMMAMBAL M.D.,D.G.O.*** Institute of Obstetrics and Gynaecology, Chennai for her guidance.

I am grateful and greatly indebted to ***Prof. Dr. RAMANI RAJENDRAN M.D., D.G.O.*** Institute of Social Obstetrics and Government Kasturba Gandhi Hospital, Chennai for her able guidance.

I extend my profound gratitude to all unit Chiefs, Registrar, Assistant Professors for their boundless affection and support for my study.

I am ever grateful for all the pregnant women who participated in this study without whom this study would not have been possible.

I thank my family & friends for their inspiration & support given to me.

CERTIFICATE

This is to certify that the dissertation entitled **"Predictor of HbA1C in abnormal OGCT in 1st and 2nd trimester and its correlation with abnormal fetal outcome in comparison with ultra sonogram"** presented herein by **Dr. R.THIRUCHELVI**, is an original work done in the Department of Obstetrics & Gynaecology, **Institute of Social Obstetrics and Government Kasthurba Gandhi Hospital**, Government Madras Medical College, Chennai, in partial fulfilment of regulations of The Tamil Nadu Dr .M. G . R. Medical University for the award of degree of M. D. (Obstetrics &Gynaecology), under my guidance and supervision during the academic period 2009-2012

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CERTIFICATE OF APPROVAL

To
Dr. R. Thiruchelvi
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Dear Dr. R. Thiruchelvi

The Institutional Ethical Committee of Madras Medical College reviewed and discussed your application for approval of the project / proposal / clinical trail entitled " Predictor of HbA1C in abnormal OGCT in 1st and 2nd trimester and its correlation with abnormal fetal outcome in comparison with ultra sonogram " No 61082010.

The following members of Ethical committee were present in the meeting held on 24.08.2010 conducted at Madras Medical College, Chennai -3.

1. Prof. S.K. Rajan, MD	– Chairperson
2. Prof. J. Mohanasundaram, MD,Ph.D,DNB Dean, Madras Medical College, Chennai -3	– Deputy Chairman
3. Prof. A. Sundaram, MD Vice Principal , MMC, Chennai -3	– Member Secretary
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6. Prof. Md. Ali, MD, DM Professor & Head ,,Dept. of MGE, MMC, Ch-3	– Member – Member
7 Prof. Shantha Ravishankar, MD Professor of Neuro Pathology, MMC, Ch-3	– Member
8. Tmt. Arnold Soulina	-- Social Scientist

We approve the trail to be conducted in its presented form.

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee

CONTENTS

INTRODUCTION

Pregnancy is an unique metabolic state in which, the mother has to provide fuel not only for herself but also for the metabolic needs of the conceptus. To accomplish this certain physiological changes. (especially is glucose metabolism) and women with preexisting aberration in insulin economics or preexisting decline in B cell function develop GDM (CURET et al).

Normal pregnancy is an insulin resistant state mediated by placental anti-insulin hormones. If the maternal pancreas cannot increase production of insulin to sustain normoglycemia despite these anti insulin hormones gestational diabetes occurs.

Gestational diabetes is a commonest metabolic disorder in pregnancy. Incidence varies between 3 to 12% depending on the sample population and diagnostic criteria used.

The name gestational diabetes mellitus was recommended by American college of obstetricians and gyenaecologists in the second international workshop conference on gestational Diabetes Mellitus in Chicago on October 25^{th} 1984, replacing the names impaired glucose tolerance in pregnancy (RUST, JAMES et al).

Gestational diabetes mellitus and other categories of glucose intolerance during pregnancy are one of the causes for increased perinatal

mortality and maternal mortality. As pregnancy proceeds the prevention of maternal complication and perinatal mortality and morbidity required continued normalization of metabolism and close surveillance of the fetal condition.

Hence it is mandatory that universal screening of all pregnant subjects must be undertaken to detect abnormal glucose tolerance during 1st and 2nd trimester for early detection and effective management.

Decreased infant, perinatal mortality and morbidity rates have followed higher detection rates and more aggressive treatment of mild carbohydrate intolerance in pregnancy.

REVIEW OF LITERATURE

HISTORY OF DIABETES:

The history of diabetes probably dates back to the beginning of human kind, encompassing centuries, generations and civilizations. There is some evidence that Hippocrates was familiar with diabetic conditions. In the writing of Hippocrates, a word is used that is translated to mean “to make water much often”.

It was areteaus of cappadocia (30-0 AD) who named diabetes which means to pass through or to siphon. He described diabetes as an affilction being a melting down of the flesh and limbs into urine.

DIABETES IN PREGNANCY:

Before 1856, there is hardly any report of pregnancy complicating diabetes. Bloh wrote that “true diabetes is inconsistent with conception”. In 1882, for the first time Duncan reported 22 pregnancies with diabetes in the literature of which 27% of mothers died at the time of labor and 22% died during the following 2 years. This trend of high maternal and fetal mortality continued until the discovery of insulin.

The advent of insulin brought about a dramatic change in the overall outlook for diabetic women and their reproductive potential. There was a dramatic fall in the maternal mortality from 45% to just over 2% shortly

after the introduction of insulin. However, the perinatal mortality did not rapidly change, but slowly decreased over time. The continuing problems are neonatal hypoglycemia, congenital malformations, infections, macrosomia and traumatic injuries to the fetus during parturition.

1. **Korean J Lab med. 2009 Apr;** 29 (2): 110-5 the relationship between the timing of gestational diabetes screening and HbA1C level and neonatal outcome.

2. **Zhong Narda xue xue ba yi xue ban 2008 Jan;** 33(1); 85.8
Glycosylated hemoglobin test in gestational abnormal glucose metabolism.

3. **Diaabetes Res din prae 2007 Sep.** 77 43) 465 to Epub 2007 mar 9 Can plasma glucose & HbA1c predict fetal growth in mothers with different glucose tolerance levels.

4. Standardisation of the reference method for the measurement of HbA1c to improve diabetes care (PDF). The association for clinical biochemistry and diabetes UK April 2008. htt pry W-WW acb. org. uk/docs/tibalc.pdf.

5. The international expert committee (2009) international expert committee report on the role of the AIC assay in the diagnosis of diabetes Diabetes care 32 (7) 1327-1334 doi :102337/dc09 9033: PMLD 19502545.

DEFINITION:

Gestational diabetes mellitus is defined as carbohydrate intolerance of variable severity with onset or first recognition during pregnancy. the

definition applies regardless of whether or not insulin is used for treatment (American Diabetes Association 1995).

Gestational diabetes mellitus affects 1-2% of all pregnancies.

MATERNAL GLUCOSE HOMEOSTASIS:

Both in early and late pregnancy the glucose concentration stays constant between 4 & 4.5 mmol/l except after meals. This degree of homeostasis is only maintained by doubling the secretion of insulin from the end of first to the third trimester of pregnancy.

Insulin resistance develops although the aetiology remains uncertain. Review by Kuhl concluded that pregnancy induced insulin resistance was likely to be a post receptor defect. This effect is mediated by increased production of one or more of free cortisol which is elevated in pregnancy associated hormones or of free cortisol which is elevated in pregnancy. Withholding food from pregnant women results in a much earlier recourse to breakdown of triglyceride, leading to increased concentration of circulatory free fatty acids and ketone bodies, described by Freinkel as “accelerated starvation.

As a result of the physiologic changes of pregnancy normal fasting blood sugar is 65+/- 9mg /dl. The mean random blood sugar level is 80+ 10 mg /d 1. Post prandial elevation never exceed 140 mg/dl.

Pathophysiology of Gestational Diabetes mellitus:

With implantation of the trophoblast the production of pregnancy related hormones begins. These hormones immediately alter the metabolism of nutrients to shift the priority of metabolic products towards the growing fetus. A buffering mechanism must be initiated early in pregnancy to prevent the mother from suffering deleterious hypoglycemia between feedings as her reserve continue to flow to her unborn child.

Human chorionic gonadotrophin, the initial hormone secreted by the syncytiotrophoblast in early pregnancy does not have an effect on glucose homeostasis.

Estrogens which rise within 35 days of conception have weak antiinsulin properties and also have a relative diabetogenic potency. As cortisol binding globulin is increased, the maternal adrenal gland secretes more cortisol resulting in an increase in the level of free cortisol. Hypercortisolemia causes maternal insulin resistance, delayed glucose clearances, and thus more available glucose for fetal use. The appearance of cortisol appears timed to contribute glucose to the rising demands for fetal fuels. The glucose tolerance is an advantage at this point in pregnancy.

Prolactin and human placental lactogen have an effect on glucose metabolism. The effect of human placental lactogen on fat and carbohydrate metabolism are similar to those following treatment on growth hormone.

There is inhibition of peripheral glucose uptake and stimulation of insulin release.

Thus the hormonal changes early in pregnancy can be viewed as a serial rise in hormones intended to maintain a constant glucose supply to the fetus. As fetal requirements increase, the gluconeogenic properties and the concentration of hormones rise. If the maternal pancreas cannot keep up with the insulin resistance produced by these hormones, blood glucose level rise which leads to gestational diabetes mellitus.

Classifiation:

Classification of GDM is very essential in order to diagnose the severity of the condition and to plan the management for achieving euglycemia and for the assessment of prognosis of the mother and the fetus.

National Diabetes Data group classification of DM:

1).Type – I DM (IDDM) - Juvenile onset DM Ketosis prone DM

2).Type – II DM (NIDDM) - Brittle DM

Adult onset DM

Maturity onset DM

Ketosis resistant DM

Stable DM

3).Type III (GCI) - Gestational carbohydrate intolerance.

Priscilla White's classification:

1. GDM - Discovered during pregnancy, glycemia may or may not be maintained by diet alone. Insulin may be required.
2. Class-A - Discovered before pregnancy, controlled with diet alone any duration or any age of onset.
3. Class-B - Age of onset 20 yrs. (or) older or duration < 10 years.
4. Class-C - Age of onset 10 to 19 years or duration 10-19 years.
5. Class-D - Age of onset under 10 years, duration over 20 years, background retinopathy.
6. Class-R - Proliferative retinopathy, vitreous haemorrhage.
7. Class-F - Nephropathy (over 500 mg per day proteinuria)
8. Class-R F - Both R + F
9. Class- H - Arterio Sclerotic heart disease clinically evident.
10. Class –T - Prior renal transplant.

American college of obstetricians & gynecologist classification, 1986 revised in 1994 Gestational Diabetes

Class	Fasting Plasma glucose mg/dl	Post Prandial Plasma glucose mg/dl
A1	<105	or <120
A2	<105	or <120

Pregestational Diabetes

Class	Age of onset	Duration (years)	Vascular disease	Therapy
A	Any	Any	None	Diet only
B	Over 20	<10	None	Insulin
C	10-19	10-19	None	Insulin
D	Before 10	>20	Benign Retinopathy	Insulin
F	Any	Any	nephropathy	Insulin
R	Any	Any	Proliferative Retinopathy	Insulin
H	Any	Any	Heart disease	Insulin

Epidemiology of GDM:

Marked variation has been reported in the prevalence of GDM world wide ranging from 2.1% (Korea) to 15%.

Author (years)	**Subjects**	**GDM prevalence%**
Biescheli etal (1991)	Vietnam Chinese Africa Indian subcontinent	7.3 13.9 9.4 15
Ranchod et al	Indian	3.8
Samanta et al	Indian	0.18
Dorrhorst et al	Indian	4.4
Agarwall et al	North Indra	1.88
Ramachandran et al	South Indian	0.56

Effects of GDM on Mother:

Preeclampsia:

Affects 10 to 25% of all pregnant diabetic patients

Infection:

High incidence of chorioamnionints and postpartum endometritis

Postpartum bleeding:

High incidence caused by exaggerated uterine distension

Caesarean section: High incidence in pregnant diabetic patients.

Effects of GDM on fetus:

The most common complication is macrosomia, which may affect upto 40% of babies, whose mothers have gestational diabetes. Common problems of infants of diabetic mother are hyperblirubinemia, hypoglycemia, hypocalcemia and hyperviscosity syndrome. Finally the incidence of fetal death and still birth is higher in gestational diabetic mother than in non-diabetic population.

Screening in pregnancy:

Need for screening:

1. The great majority of women with mild degree of carbohydrate intolerance during pregnancy do not have signs & symptoms.
2. Routine blood test and urine test are not reliable to diagnose GDM

3. Carbohydrate intolerance during pregnancy causes significant increase in fetal morbidity and mortality and maternal morbidity.
4. Early diagnosis and treatment can prevent fetal wastage.

Population to be screened:

There are two schools of thought

1. Universal screening
2. High-risk screening

Risk factor for screening:

1. Obesity or BMI > 27 before pregnancy
2. Age more than 30 years
3. Family H/O DM
4. History of delivery of large infant >4 kg.
5. Previous history of stillbirth baby with congenital anomalies unexplained perinatal and neonatal death.
6. H/O GDM is previous pregnancy
7. H/O recurrent UTI, moniliasis
8. Presence of polyhydramnios and pregnancy induced hypertension.

35-50% of women with GDM will not have any risk factors. Hence in third international workshop conference on GDM, American Diabetology association recommended universal screening for all antenatal pregnant women.

Low risk:

- Member of ethnic group with low prevalence of gestatonial diabetes.
- No known diabetes in first degree relatives
- Age less than 25 years.
- No H/O abnormal glucose metabolism
- No H/O poor obstetrical outcome

Average Risk:

Perform blood glucose testing at 24-28 weeks. Average risk includes women of Hispanic, African, Native American South or East Asian origin.

High Risk:

Perform blood glucose testing as soon as possible. If gestastional diabetes is not diagnosed blood glucose testing should be repeated at 24-28 weeks or at anytime a patient has symptoms or signs suggestive of hyperglycemia

- Marked obesity
- Strong family H/o type II diabetes
- Prior gestational diabetes
- Glucosuria

Criteria for an ideal screening test:

The test must detect the disease in a stage where early treatment will provide superior prognosis than diagnosing in late stage.

- Must be simple to use.
- Should be easy to perform in general population .
- Test must be cost effective.
- Sensitive enough to diagnose all the people, who are having the disease.
- Specific enough to exclude people who are not having the disease.

1. Methods of Screening:

a. Glucose challenge test:

This test was adopted by 0' Sullivan in 1973. In these test patients was given 50g of glucose in 200ml of water irrespective of the last meal. Venous blood was drawn 1 hour after drinking glucose and plasma blood glucose level estimated by Somogyi - Nelson method. The recommended threshold is 140 mg/d1.

Sensitivity 80%

Specific city 90%

b. 2 Hour postprandial test:

Patient was given 100mg of carbohydrate meal and various blood drawn after 2 hrs and blood glucose estimation done. The normal value is <120 mg/d1.

c. Random blood sugar estimation:

Tornkid and MC dongall (1981) conducted a study for screening of antenatal women to diagnose GDM by dividing them into two groups within two hours of last meal or after 2 hours of last meal. The reference range was

115 mg/d1 within 2 hrs of meal

105 /d1 after 2 hrs of meal

Stangenber et al 1985, Nasrat et al 1988 suggested that RBS is insensitive for the identification of GDM.

d. Fasting blood sugar:

Normally in pregnant women fasting blood sugar will be 70 to 90 mg/d1. Value >105mg/d1 suggest glucose intolerance. Only one third of GDM patient will have fasting hyperglycemia. If we take FBS as a screening test, other 2/3rd of GDM patients will not be diagnosed (METZGER BE et al, 1992).

e. WHO testing:

According to WHO expert committee 75g of oral glucose load given (patient need not be in fasting) and single plasma glucose value estimation

at 2 hrs. If glucose value is more than 140 mg% a 3 hr OGTT performed. A 75 gm 2 hrs screening test seems to be as useful in detecting GDM and is more suited to crowded outpatient clinics because its utility to detect GDM is not seriously affected by small variation in blood sampling time.

f. Seshiah spot test:

Seshiah et al (1984) used a spot with reference to a previous meal and chose the top one percentile for full OGTT. The critical spot test glucose values are 85 95 105 105 95 90 mg at 30, 60, 90, 120, 150, 180 minutes respectively in relation to the last meal.

2. Gycosylated Haemoglobin and other proteins:

Glycosylated haemoglobin and other protein have been proposed as a screening test for gestational diabetes. In HbA1C-a glucose molecule is attached to the N –terminal group of side chain of haemoglobin by non enzymatic reaction and this attachment depend on the concentration of sugar in the blood stream over prolonged period of time. Glycosylation is slow and reversible until the death of RBC's.

So glycosylation in RBCs directly proportional to the amount of glucose over a period of time, this will reflect the blood sugar control past 2 to 3 months.

Glycosylated haemoglobin was measured by a modified chemical method of fluckiger and weterhalter or micro column chromatography.

An on-chip electrochemical flow immunoassay system for the detection of hemoglobin A1C (HbA1C) was developed using anti-human hemoglobin (Hb) IgG labeled with ferrocene monocarboxylic acid (Fc–COOH) and boronate-affinity chromatography.

Drawback:

GDM however may not present with the same constant elevation of blood sugar levels as in non pregnant states. Gravid women with GDM have fasting blood sugar concentration that are low, because of increased erythropoiesis, red blood cells are younger in pregnancy, haemoglobin is less glycated and hormonal milieu changing rapidly from relative insulin sensitivity to that of insulin resistance as the pregnancy progresses.

Artal et al and course et al investigated glycosylated haemoglobin as a screening test and found to have very low sensitivity and specificity. Its measurement shows better correlation to the degree of glycemic control in the past 2 to 3 months.

Glycosylated HbA1c is often elevated in diabetes and the magnitude of the elevation correlates inversely with the degree of long term control of plasma concentration.

HbA1C:

Mean HbA1c levels are closely correlated to all meal related glucose measurements during pregnancy. It is therefore a reliable indicator of overall glycemic control among patients with diabetes during pregnancy. (Gandhi RA, BrownJ, Simma).

If the screening test for gestational DM was delayed, HbA1C level and the risk for LGA seemed to be higher, so it may be necessary to screen GDM no later than 24^{th} week of pregnancy. (Choi yJ, Kahng J)

Shah et al measured HbA1C using ion exchange chromatography and applied NDDG criteria for GDM in a group of patients with risk factor for it. This test is mainly used to predict the risk of embryopathy. HbA1C >8.5% is associated with 20-25% probability of fetal developmental abnormalities. When the value is normal the probability of major malformation is <2%. These data do not support the use of HbA1C as a screening test for GDM.

3.Serum Fructosamine test:

Fructosamine assay was proposed as a screening test for the identification of women at risk for diabetes. Fructosamine is associated with glycemic control over the previous 1 to 3 weeks, so it can be used as an appropriate maker for GDM. However this test is less sensitive than glucose challenge test (SHAH BD, COHEN AW et al) 1982.

The values of serum fructosamine are it expressed as follows:

Good diabetic control <300 mol/1

Satisfactory control 300 – 400 mol/l

Poor control >400mol/l

4.Urine sugar Test:

Glycosuria is a commonly employed screening test for detection of glucose intolerance. But during pregnancy the renal threshold for glucose is often lowered due to an eight fold increase in golmerular filtration of glucose and an intermittent tubular defect in glucose reabsorption.

This had led to the observation of long Hint (1923) that glycosuria following oral glucose load in a woman who has missed her period can be used as a test for pregnancy (federsen et al) An awareness of this fact can lead to an under diagnosis of glucose intolerance during pregnancy, while a lack of it will result in over diagnosis.

However the specificity of glucosura can be increased by defining significant glucosuria which occurs in second fasting morning specimen of urine (SUTTERCAND HWSTOWERS IM et al 1970) Pregnant women with renal glycosura are at high risk of premature delivery (25%) and fetal macrosomia 7% (CHEN et al)

Diagnostic tests for GDM:

1.100g 3 hour oral glucose tolerance test

2.75g 2 hr. oral glucose tolerance test

3. IV glucose tolerance test

Oral glucose tolerance test:

Patient should be instructed to have unrestricted diet (atleast 150 gm carbohydrate per day) and unrestricted physical activity for 3 days before the test and advised to come after an overnight fasting. This will minimize the false positive results (Journal OBS and gynae vol 70 NO-1) After taking blood for FBS. Patient is advised to drink glucose water (100 grams in 300 ml of water) Hourly blood sample should be taken for 3 hour.

GDM is diagnosed if two or more value are abnormal. If one value is abnormal, the patient cannot be diagnosed as having GDM, but they are prone for complications like macrosomia and preeclampsia.

Proposed criteria for OGTT during pregnancy

Time	Osullivan Mahan (Blood)	NDDG(1979) Plasma	Carpenter and counstan (1982)	Marketz et al 1980
F	90	105	95	105
1 hr	165	190	180	185
2 hr	143	165	159	140
3 hr	127	145	140	125 (with 75 gms glucose)

Patient in high risk group should have GTT done in early pregnancy as possible and if they have a normal glucose tolerance, the test should be repeated at 30 weeks gestation. At this gestational age the diabetogenic effect of Pregnancy is near its peak and the chances of a positive result from GTT are high. OGTT is borderline a repeat examination to confirm the diagnosis is made.

In 0'sullivan studies whole blood was tested using somogyi nelson method for measuring reducing substance. Patient with glucose tolerance test results fall between carpenter and coustan and NDDG criteria have the same probability of insulin treatment (26%) as those meeting NDDG criteria (30%) and the same probability of macrosomia.

WHO GTT:

In contrast to north America, much of the rest of world uses 75 g oral GTT during pregnancy, according to criteria adopted by world health organization.

Time	Normal	DM	GDM (Mg/d1)
Fasting 2 hrs after glucose	<110 <160	<140mg% >200mg%	<140 <140-200

Intermediate readings are classified as impaired glucose tolerance. The diagnostic criteria for diabetes in pregnancy are more stringent than

those recommended for non pregnant subjects and pregnant women with IGT should be treated as diabetes.

IVGTT:

It was adapted for use in pregnancy by silver stone et al. IVGTT offer some theoretic advantages over oral GTT. The k value allows easier analysis of glucose tolerance data and is in most circumstances independent of the method of glucose determination and is unaffected by variation in gastric emptying.

If an intravenous GTT is to be performed 0.5g of glucose / kg of ideal body weight is administered iv in 2 min or less blood glucose determination are made before the infection and at 10min interval for the following hour. These 6 plasma glucose determination are used to construct a graph. The time taken for the blood glucoses live to fall to half of its value (t ½) is used to calculate the absolute glucose disappearance rate (k) k value is calculated using the formula K=0.693 /t ½ x 100 K value of < 1.5 indicates abnormal glucose tolerance.

The lower limit of normal for k in the first trimester was 137 in the second trimester 1.18 and in the third trimester 1.13 valuve below this level were regarded as abnormal.

The oral test is more practical for out patients and is better at estimating the efficiency of glucose disposed in patient with mild

abnormalities of glucose tolerance. In addition intestinal factors appears to have an effect on normal insulin response and oral glucose ingestion represents the normal route for carbohydrate absorption. It should be pointed out that the IVGTT is useful in patients with gastrointestirial that may make the result of their oral test misleading.

Abnormal glucose tolerance during pregnancy has been associated with poor fetal outcome with identification and treatment of the same. Reliance upon risk factors for screening has been found to be unsatisfactory as the chances of missing a diagnose is around 50%.

Hence it is concluded that universal screening of all pregnant subjects must be undertaken. Irrespective of the nature of AGT subjects must be followed up till delivery, post partum GTT must be done to recognise true DM and AGT can recur during subsequent pregnancies.

In my study I have chosen HbA1c as a predictor of abnormal OGTT in Ist and 2^{nd} trimester and its correlation with abnormal fetal outcome.

Adverse effects of Diabetes:

Maternal complications:

Diabetic complication includes hypoglycemia, diabetic ketoacidosis, retinopathy, neuropathy, nephropathy and heart diseases. Comparison of specific compication in diabetic and non diabetic pregnancies as per various studies given below:

Complication	Diabetic	Control	Reference
PIH	16	7.7	Rosenn et al
Hypertension	2.5	0.3	Suhonens Terano
Hydramnios	2	0.7	Gotoman et al
Pyelonephritis	3.6	1.4	Rosenn et al
Preterm	31	20	Mimouni et al
Preterm delivery	24.6	6	Hansenc person et al
Caesarean section	45.2	12	Hanson & person
Post caesarean section infection	13.4	3.2	Diamond et al

MATENAL MORTALITY:

The composite maternal mortality rate of 0.14% was reported. To minimize the matemal mortality in addition to medical and obstetric case given antenatally. More comprehensive preconception medical evaluation and patient education are imperative.

FETAL COMPLICATIONS:

Fetus born to diabetic mother have a wide range of structural and biochemical abnormalities that can be reduced or eliminated by improved control of maternal glucose metabolism.

Fetal complications are macrosomia, congenital malformation, respiratory distress syndrome, hypoglycemia, placental immaturity and infarcts, umbilical cord edema and single umbilical artery, polycythemia / hyperviscosity syndrome, hyperbilirubinemia ,cardiomyopathy, small left colon syndrome.

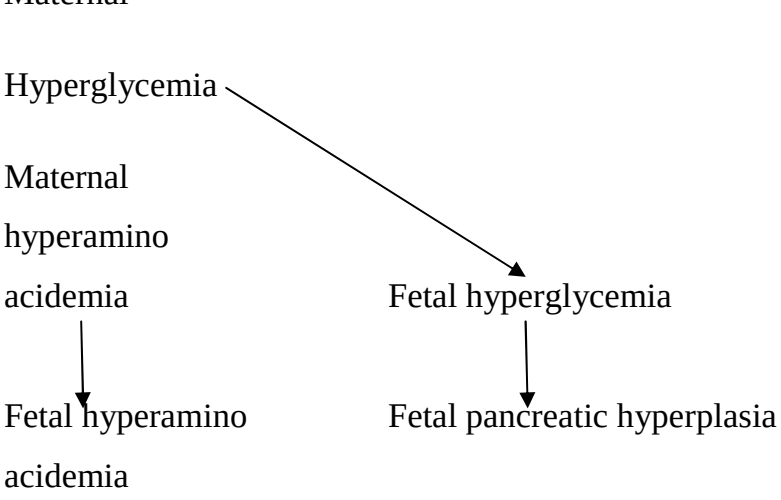
Fetal Evaluation:

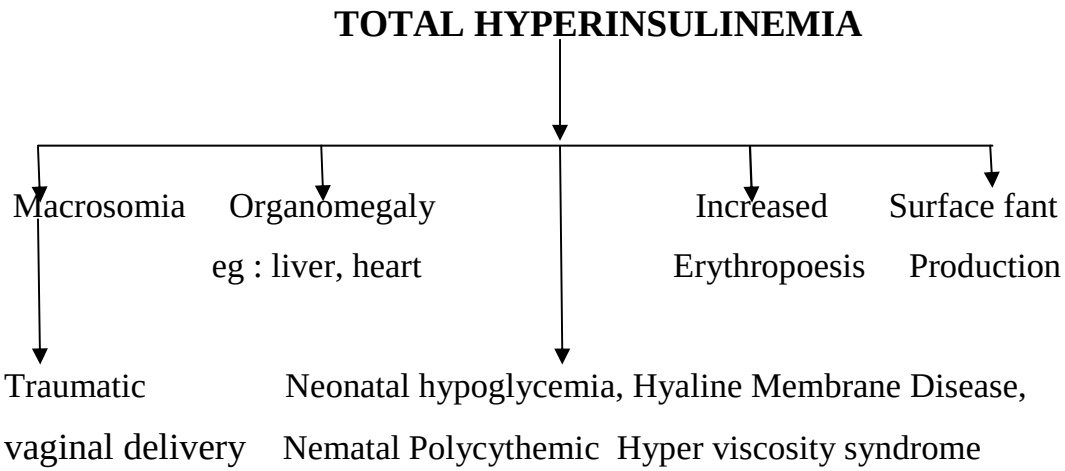
The perinatal mortality due to GDM has diminished markedly with more thorough screening programmes and intense surveillance of glycemia antepartum monitoring. Antepartum surveillance with weekly, nonstresstest and blood glucose monitoring to maintain fasting plasma glucose below 150 mg/d1 helps in diminishing perinatal mortality. The most important fetal complications are macrosomia, congenital malformation respiratory distress syndrome and sudden intrautenine death.

Macrosomia:

Macrosomia has been variously defined as birth weight greater than 4000 to 4500 g as well as large for gestational age with birth weight above 90^{th} percentile for population specific and sex specific growth curves, which complicates as many as fifty percent of pregnancies in women with GDM (MEFARLAND MB, LANGER O et, al, 2000).

PEDERSON HYPOTHESIS:





Perinatal mortality rate of 0.49% and the perinatal morbidity of 11.4% in infants weighing more than 4000 gm is five times more than of non macrosomic pregnancies.

PRENATAL DIAGNOSIS OF MACROSOMIA:

If the abdominal circumference measurement at 28-29 weeks of gestation by ultrasonogram is one centimeter or above the mean value at that stage of pregnancy, there is 77% likely hood of fetal macrosomia. If the EFW is above 90^{th} percentile macrosomia can occur in 74% of cases. However, the head circumference and BPD measurements were less predictive of macrosomia.

Predictors of shoulder dystocia:

One of the avoidable complication of macrosomia is shoulder dystocia. Modanlou et al have shown that an ultrasound derived chest to head circumference difference of 1.6 cm or a shoulder to head circumference difference of 4.8 cm predict shoulder dystocia, especially if there is an abnormality in maternal glucose tolerance.

Management:

Obviously the optimal management approach for macrosomia is prevention. Since macrosomia depends mainly upon the post prandial glucose level we should maintain at the optimal level through appropriate management. An elective cesarean delivery should be performed for an estimated fetal weight of 4000 gm or more. Most of the trails have demonstrated that prophylactic insulin therapy can in fact reduce the incidence of macrosomia as well as perinatal mortality.

O’sullivan et al in 1966 tried prophylactic insulin. He randomized women with two groups 305 were treated with prophylactic insulin and 306 constituted as untreated control groups. A third cohort of 328 non-diabetic pregnant women served as normal controls. O’sullivan et al found that 13% of the untreated women with GDM gave birth 70 babies weighing more than 4.1 kg compared with 4% of normal gravid women and a subsequent reanalysis of the data showed the insulin treatment was associated with a significant reduction in perinatal mortality rate (O’sulvivan JB, Mahan en et al, 1974).

Respiratory Distress Syndrome:

IDM’s are prove to RDS incidence . The factors influencing are:

1. Insulin may delay the fetal lung maturation in two ways.

    a. Through direct action on type II pneumocyts via increased insulin recepors and

    b. indirectly via lung fibroblasts.

2. Hyperglycemia:

    a. Decreases bio availability of important precussors for phospholipids production and surfactant protein modification.

    b. Decreased number of type II alveolar cells.

    c. Decreased number of type II cell lamellar bodies.

    d. Decreased production of phosphotidyl choline and phosphotidyl glycerol.

3. Increased beta hydroxybutyrate inhibit surfactant protein expression in fetal lung.

4. The delayed appearance of phosphotidyl glycerol in amniotic fluid in diabetic pregnancy is due to increased fetal myoinositol occurring as a result of maternal hyperglycemia.

Congenital Anomalies in infants of Diabetic mothers:

- Skeletal and central nervous sytem
- Caudal regression syndrome

Neural tube defects

- Anencephaly

Gastrointestinal:

- Duodenal atresia
- Anorectal atresia
- Small left colon syndrome
- Single umbilical artery

Cardiac

- VSD
- Coarctation
- Cardiac septal hypertrophy
- Transposition of great vessels

Renal anomalies:

- Hydronephrosis
- Renal agenesis
- ureteral duplication

Pathogenesis:

Embryos exposed to the metabolic derangement during the period of organogenesis are at increased risk of teratogenic insult.

Glycosylation of protein and abnormal vasculare observed in hyperglycemia leads to tissue under perfusion of the Embryo resulting in consequent hypoxia, acidosis cell injury and congenital malformation. The abnormalities in yolk sac tissue which is the site of origin of epithelium of many embryonic organ lead to developmental defect in these organ.

The experimental use of several different compounds such as arachidonic acid, myoinositol and antoaxidants (HOD met al, 1986) offer promise for the fetus as prophylactie against diabetic embryopathy.

Unlike in women with overt diabetes fetal anomalies are not increased in GDM.

The issue of GDM being associated with congenital anomalies is controversial prospective studies have suggested the possibility of increased rates of malformations in offspring of women with GDM. Adarshi et al in 18 month study surveyed 113 diabetic pregnancies and found that rate of congenital malformation was 6.3 in offspring of diet controlled GDM group, compared with no malformation in offspring of women with either GDM or pregestational diabetes receiving insulin.

Congenital malformations of the fetus produce a major financial and social business to the parent and the society. Prevention to a greater extent demands adequate periconceptional counseling.

Perinatal mortality and material mortality:

Before insulin became available in 1922 pregnancy and diabetes were seen as a deadly combination.

In 1882, Mathew Deencan of Fordan reported that out of 22 pregnancies into diabetic mother 4 mothers died at the time of labour another 7 died with in 2 years and nearly half the children were either born dead (or) died shortly thereafter (DUNCAN MJ et al 18820.

In this decade after insulin discovery maternal mortality rate has been reduced drastically from 50% to 0.5%.

Perinatal outcome is mainly influenced by maternal blood glucose level, obtaining maternal euglycemic remains the yardstick in the management.

Perinatal mortality rate/maternal blood glucose:

Average blood glucose (mg/01)	<100	100-150	>150
PNMR	3.4%	16%	24%

Improved fetal surveillance neonatal intensive care and maternal metabolic control have reduced perinatal losses to 2 to 4%. These rates have seemingly plateaued because the two major causes of fetal death.

Congenital malformations and unexplained fetal death remain challenging to the medical personals (GARNER).

Summary of literature:

In any high risk pregnancy obstetric outcome is mainly influenced by maternal and perinatal outcome obstetric outcome in diabetic mothers can be improved by early detection and effective management. Hence universal screening of antenatal mother for diabetes is mandatory.

Discussion :

Gestational Diabetes Mellitus (GDM) is associated with increased perinatal and maternal mortality and morbidity, which can be plummeted by early detection through screening and effective management.

Compared to European population, prevalence of GDM has increased eleven fold in women from Indian subcontinent (DORNHURST, 1992). Various studies conducted in India showed varying prevalence depending upon the population sample and the diagnostic criteria.

Prevalence of GDM in Indian subcontinent

Authors	Year	Prevalence
Agarwal et al	1982	1.88% (North Indian)
Samanta et al	1989	0.18%
Dornhurst et al	1992	4.4%
Ramachandran et al	1994	0.56% (South Indian)
C.Battacharya et al	2001	3%
Abha Jindal et al	2001	9%
Present study		2.4%

AIM OF THE STUDY

To study the HbA1c in abnormal OGCT in 1st and 2nd trimester and its correlation with abnormal fetal outcome.

MATERIAL AND METHODS

TYPE OF STUDY: Descriptive study.

STUDY PLACE:

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY, ISO-KGH, MADRAS MEDICAL COLLEGE, CHENNAI.

At the time of first antenatal visit all pregnant women are clinically evaluated by a detailed history and a proper general, system, obstetric examination and oral glucose challenge test (OGCT). Among them 500 patients with abnormal OGCT will be enrolled for the study. These patients are analysed further by HbA1C and ultrasonogram examination during 1st and 2nd trimester. After study is completed, the various levels of HbA1C are interpreted in terms of maternal and fetal outcome. HbA1C is measured by an on-chip electrochemical flow immunoassay method.

HbA1C <6% is desirable for pregnancy. Pregnancy with HbA1C >9% is advised termination. Those with HbA1C between 6-9% is correlated with USG findings and further management decided. If no abnormality detected the mother is advised for further management regarding gestational Diabetes mellitus (GDM). All the patients will be provided with routine antenatal care till delivery.

Statistical Tools (To be included at the end of Materials and Methods)

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's chi square test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

RESULTS

A: PROFILE OF CASES STUDIED

TABLE 1: AGE DISTRIBUTION

Age group	Cases	
	No	%
< 20 years	10	2.0
20 – 24 years	188	37.6
25 – 29 years	235	47.0
30 & above	67	13.4
Total	500	100
Range	19 – 36 years	
Mean	25.7 years	
SD	3.5 years	

500 women in the age group of 19 – 36 years were included in the study. Their mean age was 25.7 years and standard deviation was 3.5 years.

CHART 1: AGE DISTRIBUTION

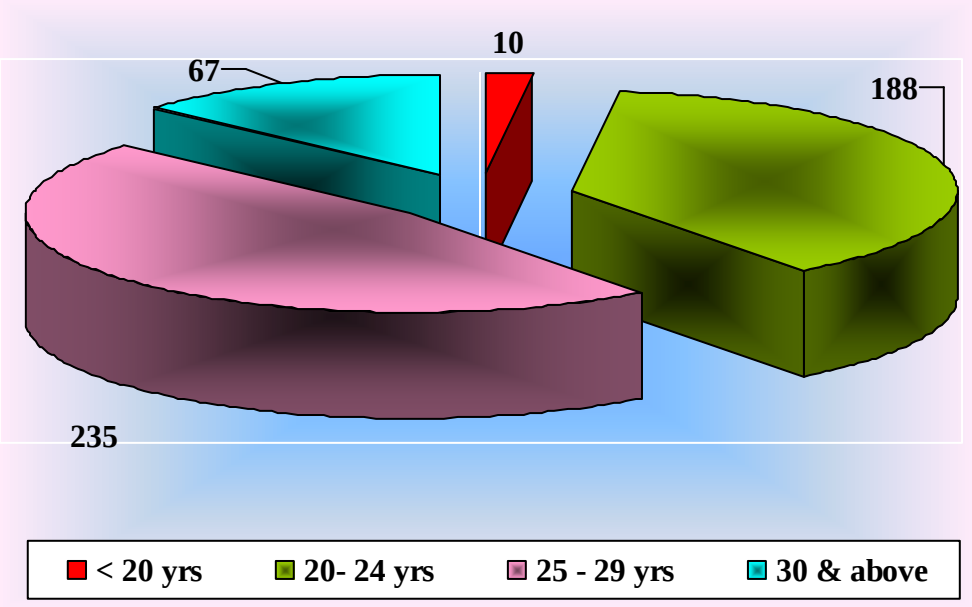


TABLE 2 : GESTATION AGE

Age group	Cases	
	No	%
< 20 weeks	155	31.0
20 – 24 weeks	265	53.0
25 – 28 weeks	80	16.0
Total	500	100
Range	13-27 weeks	
Mean	21.2 weeks	
SD	3.3 weeks	

31% of woman had a gestational age of less than 20 weeks, 53% had 20-24 weeks and 16% had 25 – 28 gestational weeks. The group had a mean of 21.2 weeks of gestation at the time of HbA1C estimation.

CHART 2: GESTATION AGE

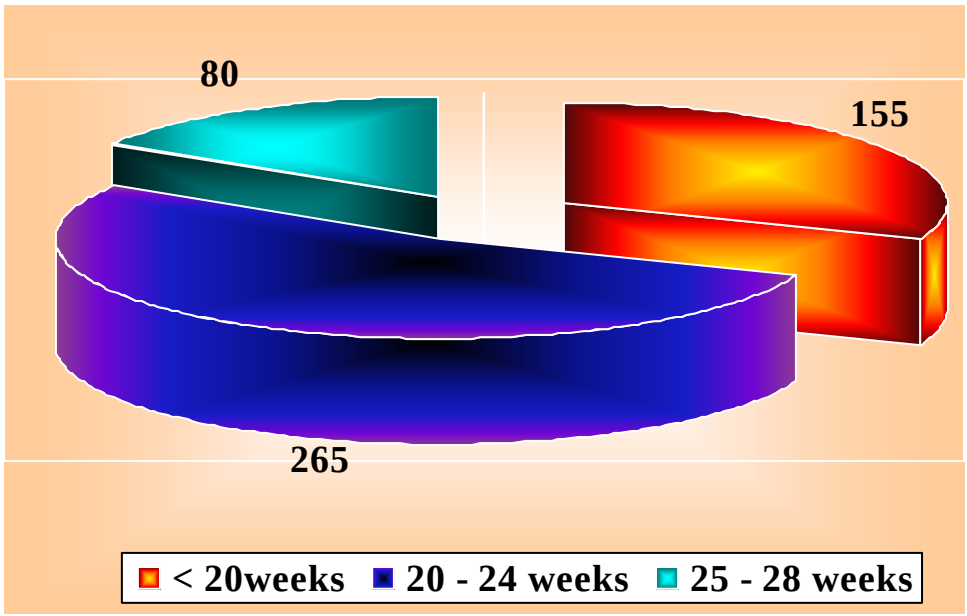


TABLE 3: OBSTETRIC CODE

Obstetric code	Cases	
	No	**%**
Primi	170	34
Multi	330	66
Total	500	100

170 women (34%) included in the study were primis and the remaining 66% were multi gravida.

CHART 3: OBSTETRIC CODE

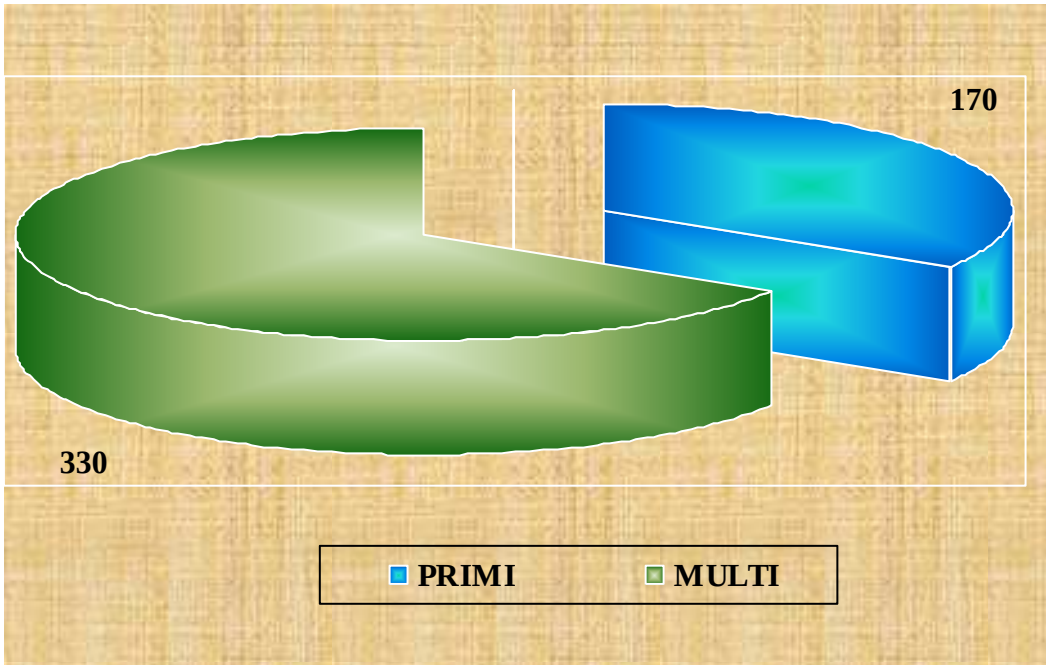


TABLE 4: USG FINDINGS

USG result	Cases	
	No	**%**
NS	496	99.2
Anomalous	4	0.8

There were 4 (0.8%) anomalous cases as per USG findings.

CHART 4: USG FINDINGS

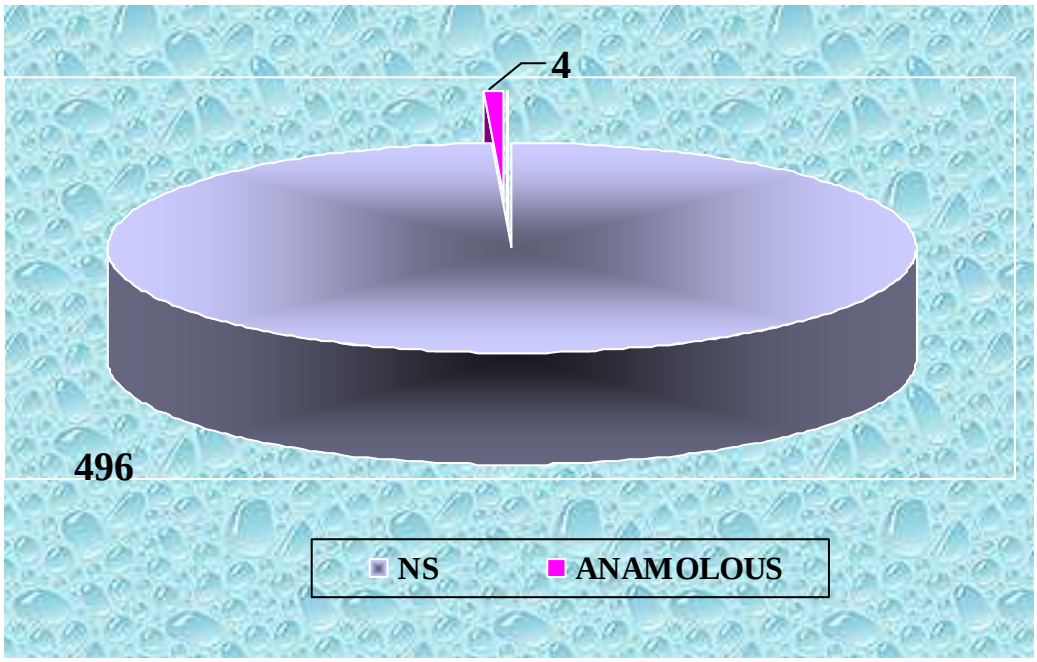


TABLE 5 : GCT

Parameter	GCT (mg/dl)
Range	140-152
Mean	145.5
SD	2.5

The study group had a GCT value of 145.5±2.5 mg/dl.

CHART 5: GCT

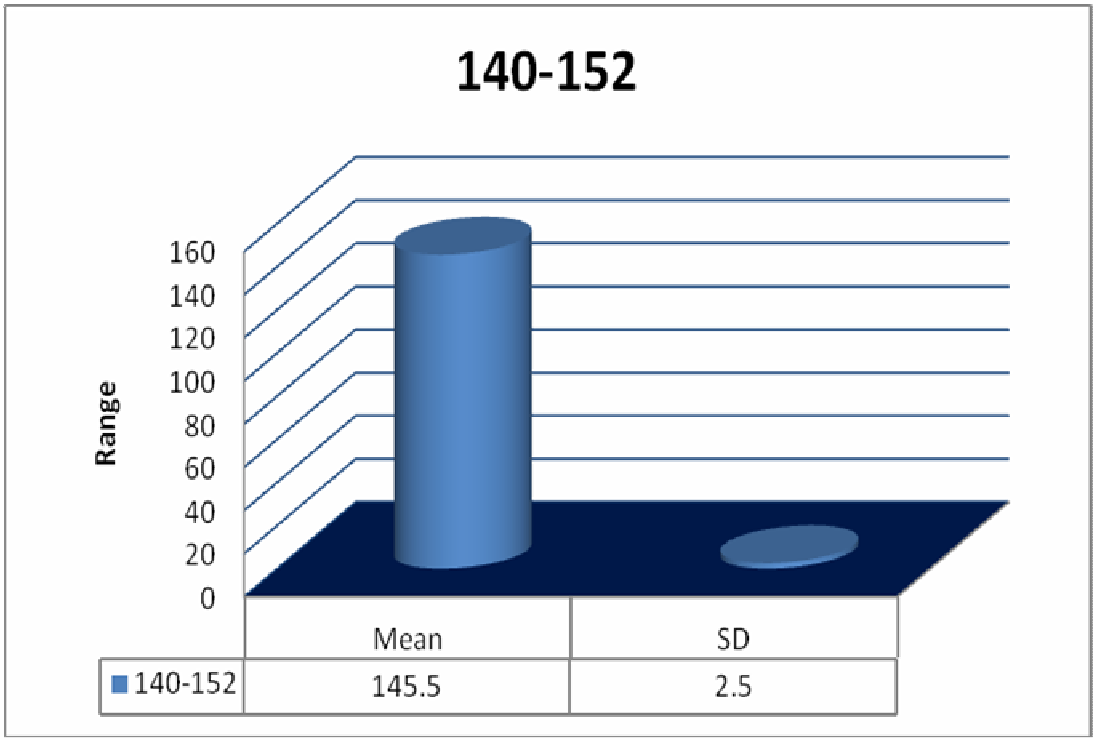

140-152
Range
160
140
120
100
80
60
40
20
0
Mean
SD
140-152
145.5
2.5

TABLE 6: HbA1C %

HbA1C	Cases	
	No	**%**
Normal ($\leq$ 6.5%)	493	98.6
Abnormal (> 8.5%)	7	1.4
Total	500	100
Range	5.1% - 7.7%	
Mean	5.65%	
S.D.	0.33 %	

Among the 500 women studied only 7 mothers (1.4%) had an abnormal HbA1C value. The group had HbA1C value of 5.65 $\pm$ 0.33%.

CHART 6: HbA1C %

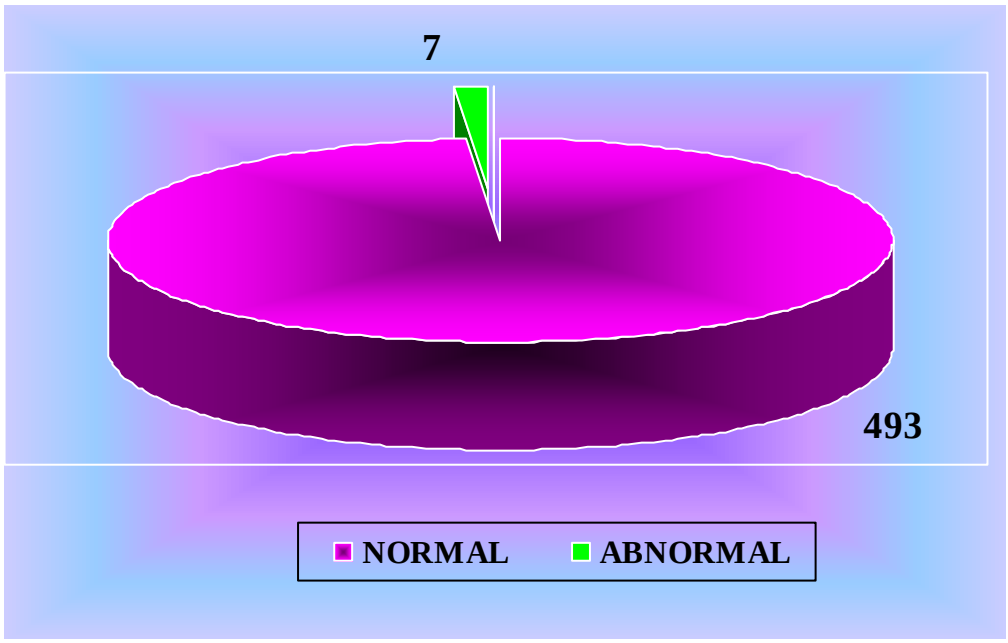
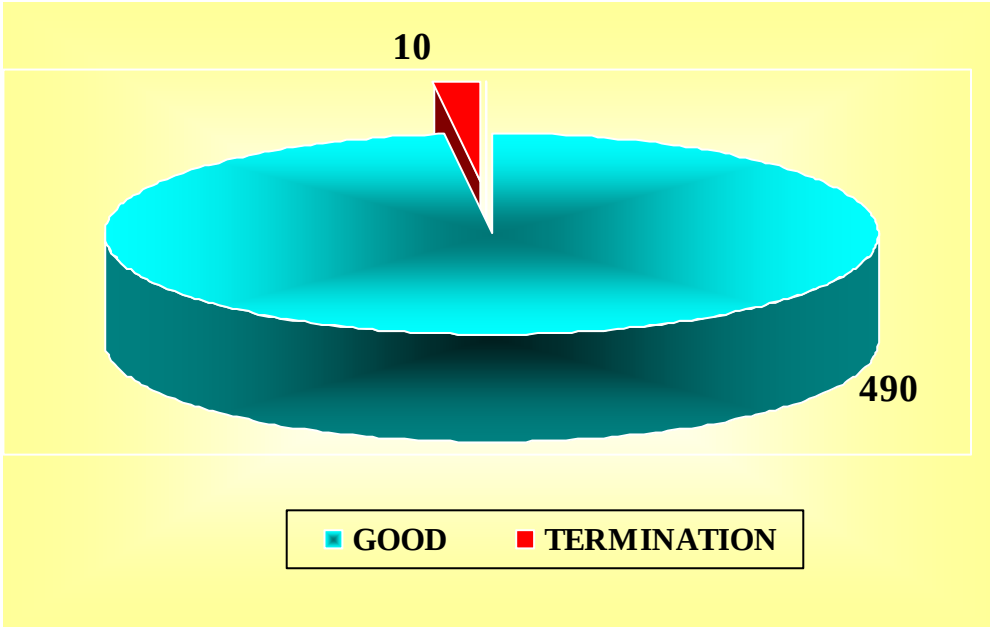


TABLE 7: FETAL OUTCOME

Fetal outcome	**Cases**	
	No	**%**
Good	490	98
Termination	10	2
Total	500	100

Among the women studied, 98% had good fetal outcome.

CHART 7: FETAL OUTCOME



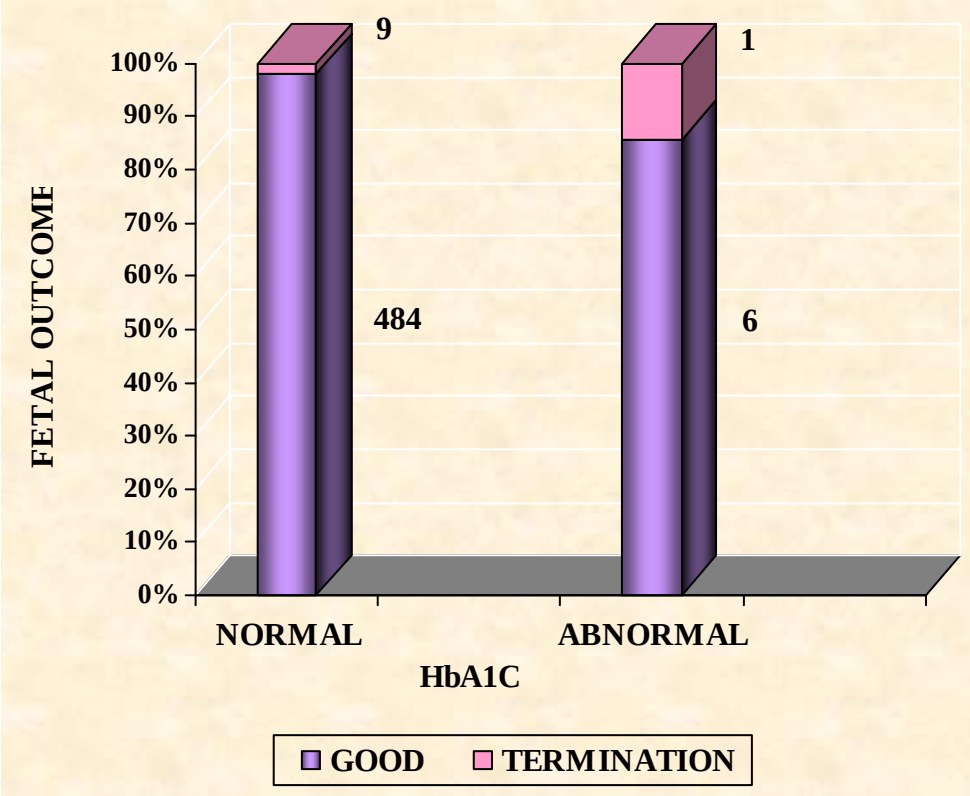
B: RELATIONSHIP BETWEEN HBA1C AND FETAL OUTCOME

TABLE 8: HbA1C AND FETAL OUTCOME

HbA1C	No.of cases	Fetal outcome			
		Good		Termination	
		No	%	No	%
Normal	493	484	98.2	9	1.8
Abnormal	7	6	85.7	1	14.3
HbA1C% Mean SD		5.64 0.32		5.91 0.75	
'p'		0.1326 Not significant			

Among mothers with normal HbA1C values, there were 9 terminations and in mothers with abnormal HbA1C one termination. The mean HbA1C values were 5.64 for those with good fetal outcome and 5.91 for those with abnormal outcome. These differences were not statistically significant ($p = 0.1326$).

CHART 8: HbA1C AND FETAL OUTCOME



C : RELATIONSHIP BETWEEN OTHER VARIABLES AND FETAL OUTCOME

TABLE 9: AGE AND FETAL OUTCOME

Fetal outcome	**Age in years**	
	Mean	**SD**
Good	25.7	3.5
Termination	25.1	3.8
'p'	0.5641 Not significant	

Age of mothers did not have significant relationship with fetal outcome ($p > 0.05$).

CHART 9: AGE AND FETAL OUTCOME

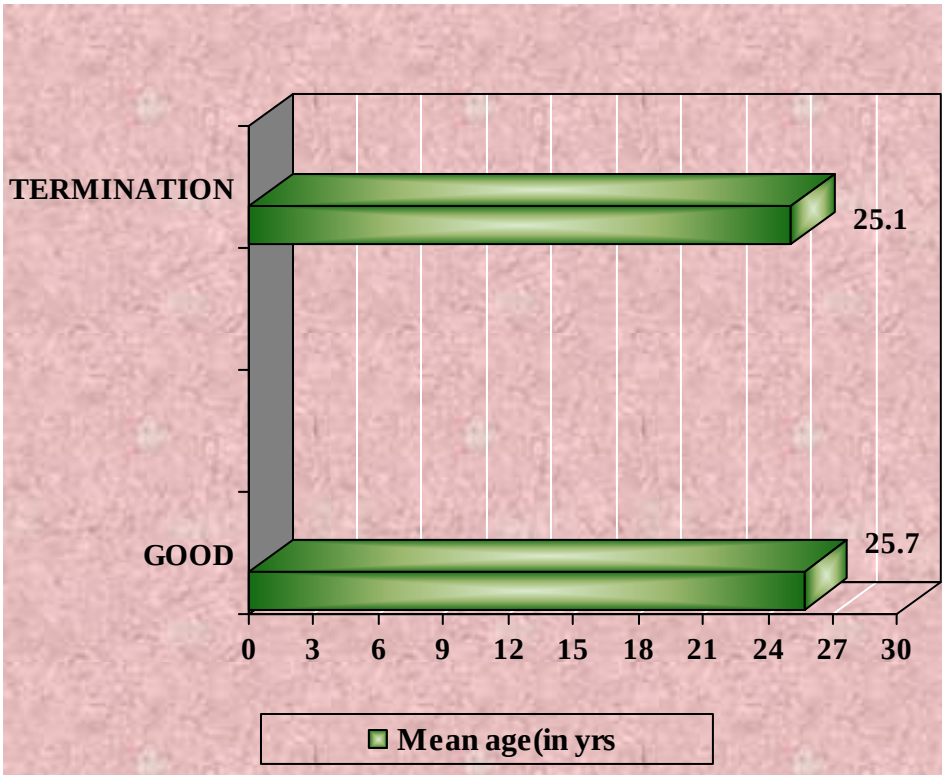


TABLE 10: OBSTETRIC CODE AND FETAL OUTCOME

Obstetric code	No.of cases	Fetal outcome			
		Good		Termination	
		No	%	No	%
Primi	170	166	97.6	4	2.4
Multi	330	324	98.2	6	1.8
'p'		0.459 Not Significant			

Among primis, 97.6% had good fetal outcome and among multis 98.2% had good outcome. This relationship was not statistically significant (p = 0.459).

CHART 10: OBSTETRIC CODE AND FETAL OUTCOME

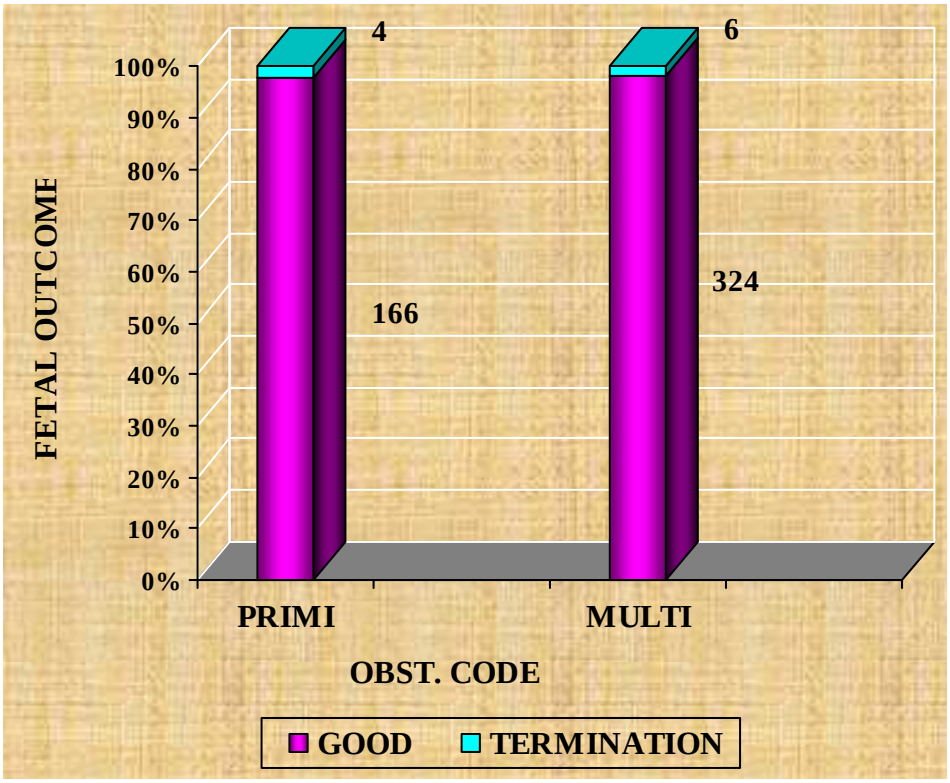
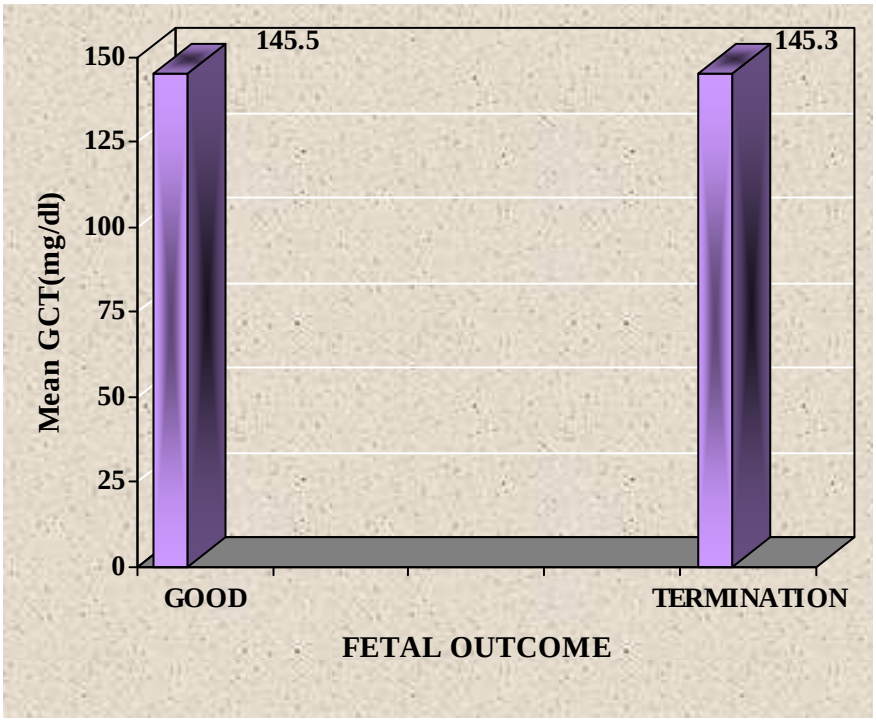


TABLE 11: GCT AND FETAL OUTCOME

Fetal outcome	**GCT (mg/dl)**	
	Mean	**SD**
Good	145.5	2.5
Termination	145.3	3.1
'p'	0.5696 Not Significant	

Mothers with poor fetal outcome had GCT of 145.3 $\pm$3.1 and mothers with good outcome had GCT of 145.5$\pm$ 2.5). The relationship was not statistically significant.

CHART 11: GCT AND FETAL OUTCOME



DISCUSSION

Elevated HbA1C is associated with increased adverse pregnancy outcomes (eg abortion , stillbirth, and congenital abnormalities. However, a level of AIC below which further improvement will carry no benefit for the fetus has not been defined.

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Glycosylated hemoglobin test in gestational abnormal glucose metabolism.

According to several studies there seems to be a family broad range (up to around 10SDS above the reference level) of acceptable metabolic control around conception over which the risk of acceptable AIC values. suhonen et al found that even a slightly raised AIC level was positively associated with an increased risk of major congenital abnormalities. We likewise found no indication of such an AIC threshold level, but our risk estimates were imprecise due to a small number of adverse outcomes. Low level of AIC can only be achieved at the cost of an increased risk of hypoglycemia episodes, which are often feared by both patients and doctors, therefore it is of interest to establish whatever there exists an AIC threshold below which further reduction will not improve fetal prognosis.

SUMMARY

In our present study 500 antenatal women with abnormal glucose challenge test in first and second trimester had been selected and subjected to HbA1C estimation.

Among the 500 women studied only 7 mothers had an abnormal HbA1C value 6 had good fetal outcome which accounts for 85.7% and 1 had abnormal fetal outcome which accounts for 14.3%

The mean HbA1C values were 5.64 for those with good fetal outcome and 5.91 for those with abnormal outcome these differences were not statically significant.

CONCLUSION

In our present study 500 antenatal women with abnormal glucose challenge test in first and second trimester had been selected and subjected to HBA1C estimation.

The study revealed HbA1C is not a good predictor for GDM with adverse fetal outcomes.

Hence the study HbA1C is not a marker for GDM with congential anomalies.

PROFORMA

- NAME
- AGE
- SOCIO ECONOMIC STATUS
- ADDRESS
- GRAVIDITY
- LMP
- EDD
- MENSTRUAL H/O
- MARITAL H/O
- OBSTETRIC H/O
- PAST H/0

GENERAL EXAMINATION

- PULSE RATE
- BLOOD PRESSURE
- ANAEMIA
- PEDAL EDEMA
- BREAST
- THYROID
- SPINE
- PER ABDOMEN
- PERVAGINUM

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MASTER CHART

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
1	LAKSHMI	25	3014	G2P1L1	13	145	6	NS	GOOD
2	AMBIKA	24	3088	PRIMI	20	148	5.2	NS	GOOD
3	GRACE	22	3725	PRIMI	21	143	6	NS.	GOOD
4	SHEEBA	28	3645	G3A2	24	148	5.6	NS	GOOD
5	LALITHA	32	3212	G2P1L0	23	149	5.2	NS	GOOD
6	LILLY	29	3423	PRIMI	26	145	5.7	NS	GOOD
7	JEBA	21	7625	G2A1	24	142	5.3	NS	GOOD
8	RAMYA	24	3545	PRIMI	23	146	5.4	NS	GOOD
9	FATHIMA	23	3654	G2P1L0	24	149	5.7	NS	GOOD
10	PRIYA	24	3767	G3P2L1	26	145	5.8	NS	GOOD
11	DEVI	27	3645	PRIMI	22	143	5.4	ANOMALOUS	TERMINATION
12	ANNAM	21	3256	G2P1L1	26	148	5.6	NS	GOOD
13	VEMBU	25	3897	G2A1	25	143	5.7	NS	GOOD
14	KANIKA	21	3745	PRIMI	24	150	5.2	NS	GOOD
15	VANI	32	3264	PRIMI	25	151	6.7	NS	GOOD
16	NITHIYA	36	2546	G2P1L1	24	143	5.7	NS	GOOD
17	RAJI	26	3612	PRIMI	26	145	5.9	NS	GOOD
18	DIVIYA	19	3765	G2A1	24	142	5.3	NS	GOOD
19	PUSHPA	23	3243	G3P2L2	23	147	6.2	NS	GOOD
20	USHA	27	3906	PRIMI	26	148	5.7	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
21	MARIAM	24	3712	G2P1L1	25	147	5.1	NS	GOOD
22	ANITHA	27	3456	G2P1L1	24	146	5.6	NS	GOOD
23	AMBIKA	21	2434	PRIMI	26	143	5.2	NS	GOOD
24	SATHIYA	21	3762	PRIMI	21	142	5.3	NS	GOOD
25	VANI	27	3154	G3P1L1A1	23	145	5.4	NS	GOOD
26	MANJU	33	3651	G2A1	27	150	5.8	NS	GOOD
27	RAJI	22	3871	G2A1	24	143	5.4	NS	GOOD
28	KUMARI	19	3908	PRIMI	26	147	5.6	NS	GOOD
29	NATHIYA	23	3710	PRIMI	26	149	5.6	NS	GOOD
30	KOWSEI	26	3004	G2P1L1	24	148	5.3	NS	GOOD
31	KAIALB	21	3612	G4A3	23	146	5.2	NS	GOOD
32	BHARTHI	24	3817	G2A1	25	143	5.1	NS	GOOD
33	THARANI	32	3945	G3P1L1A1	25	150	5.4	NS	GOOD
34	KGUMARI	24	3732	PRIMI	24	146	5.5	NS	GOOD
35	ANITHA	21	3767	PRIMI	23	145	5.2	NS	GOOD
36	SUJI	26	3434	G3A2	24	145	5.1	NS	GOOD
37	VALI	19	3624	G2P1L1	24	143	5	NS	GOOD
38	REMYA	28	3898	G2A1	24	147	6.7	NS	GOOD
39	SHYNI	28	3909	G2P1L0	25	148	5.3	NS	GOOD
40	BLESSY	19	3709	G2P1L1	26	145	5.2	NS	GOOD
41	JAMILA	22	3203	G2A1	23	146	5.3	NS	GOOD
42	MALIKA	27	3150	PRIMI	21	146	5.4	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
43	SHANTHI	32	3265	PRIMI	27	146	5.8	NS	GOOD
44	JAYA	20	4512	G2P1L1	25	143	5.4	NS	GOOD
45	MARY	30	2369	PRIMI	21	142	5.5	NS	GOOD
46	MALI	19	5125	PRIMI	23	147	5.3	NS	GOOD
47	RAJI	22	2356	G2A1	25	146	5.6	NS	GOOD
48	SHEELA	28	2969	G2P1L1	24	149	5.6	NS	GOOD
49	RAGA	24	1569	G3P1L1A1	26	143	5.6	NS	GOOD
50	AJIM	26	2365	PRIMI	27	148	5.7	NS	GOOD
51	JAYA	27	4865	PRIMI	23	142	5.4	NS	GOOD
52	ELAVARASI	25	2314	PRIMI	25	147	5.5	NS	GOOD
53	LENI MARY	22	1856	G2A1	24	145	5.9	NS	GOOD
54	JANCY	26	5369	G2P1L1	24	145	5.8	NS	GOOD
55	ALMELU	28	4236	G3P2L2	26	146	5.6	NS	GOOD
56	SHALINI	29	2658	G2P1L1	25	145	5.2	NS	GOOD
57	RATHNA	26	2159	G2P1L1	25	147	5.2	NS	GOOD
58	SIVRANJANI	32	3698	PRIMI	26	147	5.5	NS	GOOD
59	JANCYRANI	34	2489	PRIMI	23	148	5.6	NS	GOOD
60	KALIVANI	22	2178	PRIMI	23	145	5.8	NS	GOOD
61	NANTHINI	26	2695	G2A1	24	143	5.3	NS	GOOD
62	KANMANI	29	3125	G3P1L1A1	25	145	5.1	NS	GOOD
63	SAROJA	25	3254	PRIMI	25	142	5.3	NS	GOOD
64	GAYATHRI	32	2985	G2P1L1	24	143	6	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
65	KAVITHA	24	2569	G2P1L1	26	149	5.4	NS	GOOD
66	KANIKA	29	2489	G3A2	23	147	5.4	NS	GOOD
67	CLARA	27	2658	G2A1	27	147	5.4	NS	GOOD
68	SHEELA	21	2478	PRIMI	24	145	5.3	NS	GOOD
69	THARA	23	2698	PRIMI	25	146	5.6	NS	GOOD
70	PUSPHA	21	3124	PRIMI	23	145	5.6	NS	GOOD
71	THARA	24	3276	G2P1L1	16	141	5.5	NS	GOOD
72	SOBANA	32	2145	PRIMI	24	145	5.7	NS	GOOD
73	KAVIYA	34	2458	G2A1	27	145	5.7	NS	GOOD
74	LAKSHMI	32		PRIMI	25	140	5.7	NS	GOOD
75	KIRTHEEKA	26	2698	PRIMI	25	143	5.4	NS	GOOD
76	JAYANTHI	28	2478	G2P1L1	24	148	5.3	NS	GOOD
77	SOLOKCHANA	26	3248	G2A1	24	148	5.2	NS	GOOD
78	SUGANTHI	21	3125	G3P1L1	21	149	5.2	NS	GOOD
79	MALARGODI	25	4865	G3P2L2	23	145	4.9	NS	GOOD
80	RAVATHI	26	2985	PRIMI	26	147	5.2	NS	GOOD
81	SUGANYA	23	2487	PRIMI	25	146	5.3	NS	GOOD
82	RATHIKA	24	2698	G2A1	24	143	5.4	NS	GOOD
83	SANGEETHA	21	2147	G2A1	24	148	5.4	NS	GOOD
84	CHANDRA	29	2985	G2P1L0	23	148	5.9	NS	GOOD
85	MANGALA	26	3248	PRIMI	24	148	5.6	NS	GOOD
86	MANOGARI	24	2145	G2P1L1	27	147	5.5	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
87	SUBULAKSHIMI	25	2487	G3P2L2	26	149	5.5	NS	GOOD
88	JAYALAKSHM	23	1265	PRIMI	23	146	5.4	NS	GOOD
89	RAJALAKSHMI	25	1478	G2P1P1	24	146	5.3	NS	GOOD
90	NATHIYA	29	2698	G2P1L1	24	143	5.6	NS	GOOD
91	DIVYA	21	3214	PRIMI	24	143	5.5	NS	GOOD
92	KALAYANI	26	2358	PRIMI	25	143	5.2	NS	GOOD
93	CHITHRA	32	1698	PRIMI	25	146	5.3	NS	GOOD
94	KAVITHA	34	3985	PRIMI	24	148	5.4	NS	GOOD
95	KRISHNAVENI	31	1956	G2A1	27	147	5.4	NS	GOOD
96	ANUSHYA	26	1265	G2P1L1	23	146	5.3	NS	GOOD
97	PRIYA	24	1598	G2P1L1	26	145	5.1	NS	GOOD
98	AMMU	29	2589	PRIMI	25	143	5.8	NS	GOOD
99	ANIJA	24	1325	PRIMI	25	147	5.9	NS	GOOD
100	PONMANI	27	2698	G2A1	26	147	5.2	NS	GOOD
101	SEETHA	29	2985	.PRIMI	14	142	5.1	NS	GOOD
102	RANI	25	1698	.G2P1L1.	14	I45	6.5	NS	GOOD
103	SUDHA	21	1789	PRIMI	16	141	5.7	NS	POOR
104	KAMACHI	26	2536	.G2A1.	16	143	5.9	NS	GOOD
105	ROSI	24	4289	G3P2L2	18	142	5.1	NS	GOOD
106	SANTHA	23	3269	G2P1L0	13	149	5.9	NS	GOOD
107	LAVANYA	24	2564	PRIMI	22	141	5.7	NS	GOOD
108	SWEETY	25	3254	G2P1L1	24	146	5.1	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
109	PRADEEPA	22	2541	PRIMI	18	142	5.2	NS	GOOD
110	RENUGA	25	2985	PRIMI	18	140	5.6	NS	GOOD
111	ANITHA	29	2415	G2AI	16	148	6	NS	GOOD
112	SAVEETHA	24	2365	G3P1L1A1	24	147	5.2	NS	GOOD
113	BARANI	26	2896	G2P1L1	18	143	5.9	NS.	GOOD
114	PAVITHRA	28	3256	G2P1L1	20	141	5.3	NS	GOOD
115	RAJAKUMARI	26	2487	PRIMI	14	145	5.1	NS	GOOD
116	DEEPA	32	1568	G3P2L2	19	142	6	NS	GOOD
117	MUNIYAMMA	29	1267	G3P2L2	20	141	5.8	NS	GOOD
118	ANNAMALAI	22	2359	G2P1L1	22	145	5.2	NS	GOOD
119	KASTURI	24	3256	G2P1L1	20	142	5.6	NS	GOOD
120	SELI	23	1258	G3A2	18	143	5.9	NS	GOOD
121	NARMATHA	26	1478	PRIMI	16	141	5.7	NS	GOOD
122	AMBIKA	24	2698	PRIMI	16	142	6	NS	GOOD
123	SARITHA	29	2369	G2P1L1	20	142	5.2	NS	GOOD
124	DAHNALAKSHMI	31	1498	G3P2L1	22	147	5.8	NS	GOOD
125	SATHYA	25	2541	G2P1L1	22	146	6	NS	GOOD
126	MAGESHWARI	20	3012	PRIMI	19	141	5.5	NS	GOOD
127	UMA	26	2158	PRIMI	22	144	5.5	NS	GOOD
128	VIJAYALAKSH	25	1478	G2P1L1	18	152	9.7	ANAMOLOUS	TERMINATION
129	SHYLAJA	23	2569	PRIMI	14	141	5.1	NS	GOOD
130	SRIDEVI	29	3256	G2P1L1	16	142	6	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
131	MALATH	24	3842	G2A1	16	149	5.3	NS	GOOD
132	SATHYAPRIYA	25	2147	G3P2L1	20	145	5.6	NS	GOOD
133	KOSALYA	26	2145	G3P2L2	22	146	5.8	NS	GOOD
134	MALAR	28	1698	G2P1L1	20	142	5.6	NS	GOOD
135	DEVIPRIYA	20	2589	PRIMI	24	147	5.8	NS	GOOD
136	VANI	31	1523	G4P3L1	20	143	5.9	NS	GOOD
137	INDU	32	1473	G2P1L1	22	149	5.7	NS	GOOD
138	RAGA	26	2594	G2P1L1	16	145	5.6	NS	GOOD
139	RAGAMATH NISHA	28	2386	G2P1L1	18	142	5.9	NS	GOOD
140	RASATHI	25	3086	G3A2	18	143	5.6	NS	GOOD
141	PADMA	26	2853	G2P1L1	20	146	5.8	NS	GOOD
142	BAKYA	24	2586	G2P1L1	22	143	5.5	NS	GOOD
143	PARAMESHWARI	28	2185	G3P2L2	22	144	5.6	NS	GOOD
144	VALAR	21	2954	G2P1L1	24	143	5.8	NS	GOOD
145	MURUGESHWARI	22	3258	G2A1	22	142	5.9	NS	GOOD
146	SUNDARI	25	2145	G2P1L1	20	145	5.7	NS	GOOD
147	PODUMPONA	23	2698	G2P1L1	18	149	5.5	NS	GOOD
148	MANIMEGALAI	21	2478	PRIMI	22	147	5.5	NS	GOOD
149	GANASUNDARI	22	2563	G2A1	21	145	5.6	NS	GOOD
150	UMA	23	3021	PRIMI	24	146	5.9	NS	GOOD
151	VENODINI	26	3589	G2P1L1	22	148	5.7	NS	GOOD
152	PRABA	29	2145	G3P2L2	20	146	5.6	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
153	IYAMMA	31	2658	G4P3L1	20	149	5.9	NS	GOOD
154	UNNAMALI	31	3021	G2P1L1	18	143	5.7	NS	GOOD
155	CHELLAMMA	30		G2P1L1	20	144	5.8	NS	GOOD
156	RADIKA	26	2136	G2P1L1	14	145	5.9	NS	GOOD
157	SASIKALA	29	2863	G3P2L2	20	146	5.6	NS	GOOD
158	PONGODI	24	2154	G2P1L1	16	147	5.4	NS	GOOD
159	USHA RANI	25	2031	G2P1L1	18	145	5.6	NS	GOOD
160	KAMACHI	29	3025	G3P2L1	16	148	5.6	NS	GOOD
161	ARULMOZIL	28	1203	PRIMI	18	142	5.8	NS	GOOD
162	MARVIZLI	32	2691	G2A1	20	146	5.6	NS	GOOD
163	GOMATHIDEVAKI	25	2013	G2P1L1	22	143	5.8	NS	GOOD
164	RAMANI	26	2325	G2P1L1	18	145	5.8	NS	GOOD
165	VENILA	21	2486	G2P1L1	16	148	5.6	NS	GOOD
166	MYTHILI	28	2148	G3P2L2	22	149	5.5	NS	GOOD
167	RENUKA	29	2963	G3P1L1	20	147	5.2	NS	GOOD
168	AMUTHA	21	3058	G2A1	24	141	5.1	NS	GOOD
169	KARPAKAM	24	3045	G2P1L1	16	142	5.3	NS	GOOD
170	MEEANKSHI	25	1258	G2P1L1	24	148	5.4	NS	GOOD
171	RASATHI	26	1632	G2P1L1	20	143	5.8	NS	GOOD
172	KANAMMA	28	2147	G3P2L2	22	146	5.9	NS	GOOD
173	KUMUTHA	24	2369	PRIMI	24	145	5.7	NS	GOOD
174	RANI	26	2541	PRIMI	16	145	5.6	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
175	RAMA	21	3086	PRIMI	18	143	5.5	NS	GOOD
176	KUMARI	20	2147	G2P1L1	22	141	5.3	NS	GOOD
177	MANIMEGALI	26	3621	G2P1L1	24	142	5.7	NS	GOOD
178	SIVAGAMI	21	1205	PRIMI	20	149	5.8	NS	GOOD
179	SUSILLA	25	1682	G2P1L1	16	148	5.9	NS	GOOD
180	ESWARI	26	2698	G2A1	18	147	5.5	NS	GOOD
181	THENMIZOHI	21	3258	PRIMI	18	143	5.5	NS	GOOD
182	JAMUNA	23	1258	G3P1L1	20	146	5.8	NS	GOOD
183	VASANTHI	28	2596	G2P1L1	24	142	5.6	NS	GOOD
184	SETALLA	24	3254	G2P1L1	20	145	5.8	NS	GOOD
185	VIJAYA	21	1286	PRIMI	24	150	5.6	NS	GOOD
186	HEMAVATHI	20	3256	PRIMI	16	141	5.5	NS	GOOD
187	JEROM	22	1586	G2P1L1	14	143	5.6	NS	GOOD
188	CELLIN	30	2158	G2P1L1	20	144	5.4	NS	GOOD
189	SUSEELA	31	3589	G3P2L2	22	149	5.8	NS	GOOD
190	JANAKI	26	1258	G2P1L1	18	147	5.4	NS	GOOD
191	SUDHA	25	1698	G3P2L2	24	146	5.2	NS	GOOD
192	PARVEEN	21	3258	G2A1	22	142	5.6	NS	GOOD
193	DURGADEVI	23	2145	PRIMI	20	147	5.4	NS	GOOD
194	AYSHA	28	1586	G3A2	18	146	5.7	NS	GOOD
195	INDUMATHI	26	3698	G2P1L1	16	142	5.9	NS	GOOD
196	BAKYAVATHI	24	1258	G2P1L1	18	141	5.1	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
197	VALLI	21	1263	G2P1L1	16	140	5.6	NS	GOOD
198	SALIMA	29	3248	G3P2L1	20	143	5.7	NS	GOOD
199	BOMMI	25	1526	PRIMI	22	145	5.6	NS	GOOD
200	FLORA	23	3698	G2P1L1	24	142	5.7	NS	GOOD
201	AKSHYA	31	2148	G4P2L2A1	22	146	5.9	NS	GOOD
202	JEEBA	28	1589	G2P1L1	20	147	5.3	NS	GOOD
203	BEGAM	24	2365	PRIMI	18	149	5.8	NS	GOOD
204	INDRANLEKA	25	1258	G2P1L1	16	143	5.9	NS	GOOD
205	CHANDRA	26	1693	G2P1L1	20	148	5.6	NS	GOOD
206	SHAJEETHJA	21	2485	PRIMI	16	147	5.9	NS	GOOD
207	ASLIMA	29	2569	G2P1L1	18	149	5.6	NS	GOOD
208	PANCHAYAMMA	31	3014	G3P2L2	16	143	5.9	NS	GOOD
209	NARKIESS BANU	30	2563	G2P1L1	20	148	5.6	NS	GOOD
210	MANGAYAR ARASI	23	2485	PRIMI	24	145	5.8	NS	GOOD
211	MAHALAKSHMI	33	1258	G3P2L2	18	147	5.7	NS	GOOD
212	KALPANA	29	3021	G3P2L2	16	146	5.6	NS	GOOD
213	GANDIMATHI	24	1250	G2P1L1	14	143	5.9	NS	GOOD
214	VEDAVALLI	20	1243	PRIMI	20	141	5.3	NS	GOOD
215	RASATHI	29	2635	G2P1L1	22	143	5.6	N S	GOOD
216	MUTHU	32	1258	PRIMI	18	146	5.8	NS	GOOD
217	RAJANI	26	1369	G2P1L1	18	146	5.7	NS	GOOD
218	BALA	21	2569	PRIMI	16	143	5.9	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
219	ANALAKSHMI	24	3256	G2P1L1	14	146	6	NS	GOOD
220	GIRIJA	26	3025	G2P1L1	18	149	5.1	NS	GOOD
221	JERCY	29	3058	G3P2L2	20	149	5.9	NS	GOOD
222	DAICY	25	3025	G2P1L1	22	143	5.7	NS	GOOD
223	PARIMALA	30	1526	G3P2L1	20	145	5.8	NS	GOOD
224	SUMITHRA	31	1478	G3P2L1	20	146	5.6	ANAMOLOUS	TERMINATED
225	PRAMA	21	1269	PRIMI	24	143	5.9	NS	GOOD
226	ARTHIA	26	2589	PRIMI	22	148	6	NS	GOOD
227	ABIRAMI	25	3254	G2P1L1	20	146	5.9	NS	GOOD
228	MUBINA AZIM	21	2458	G2P1L1	22	142	5.8	NS	GOOD
229	NAGALASHMI	24	1589	G2A1	24	145	5.8	NS	GOOD
230	JOTHI	26	1269	PRIMI	18	150	5.3	NS	GOOD
231	MANGAI	21	3256	PRIMI	16	146	5.8	NS	GOOD
232	MANJULA	28	1478	G2P1L1	14	145	5.7	NS	GOOD
233	GEETHA	29	3025	G2P1L1	16	147	5.6	NS	GOOD
234	PRABAVATHI	30	3069	G3P2L2	18	148	5.5	NS	GOOD
235	LAITHA	22	2589	PRIMI	20	149	5.4	NS	GOOD
236	SENBAGAM	24	3214	G2P1L1	22	147	5.7	NS	GOOD
237	ANANDHI	25	2019	G2P1L1	24	146	5.8	NS	GOOD
238	KOKILA	26	2365	G2P1L1	20	145	5.6	NS	GOOD
239	LALITHA	32	2563	G3A2	22	146	5.7	NS	GOOD
240	JASMINE	21	2369	PRIMI	20	147	5.9	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
241	SAGAYAM	25	2563	G2A1	24	149	5.9	NS	GOOD
242	TAMILSELVI	30	1698	G3P2L1	22	143	5.5	NS	GOOD
243	ELAKYA	25	2365	G2P1L1	16	144	5.7	NS	GOOD
244	BARANI	24	2147	PRIMI	18	145	5.4	NS	GOOD
245	BALA	28	2569	G3P2L1	22	145	5.6	NS	GOOD
246	PECHI	21	1258	PRIMI	20	143	5.7	NS	GOOD
247	SHARMILASENAKA	30	1369	G2P1L1	16	148	5.9	NS	GOOD
248	MUMTAJ	20	3210	G2P1L1	18	145	5.6	NS	GOOD
249	PONGAVANA	25	2147	G2A1	22	148	5.8	NS	GOOD
250	JENITHA	26	3256	G2P1L1	20	147	5.6	NS	GOOD
251	REENA	24	1528	G2A1	16	143	5.5	NS	GOOD
252	SOLOMI	25	2598	G2P1L1	14	148	5.7	NS	GOOD
253	RANI	26	2158	G2P1L1	21	147	5.8	NS	GOOD
254	REVATHI	26	1569	G2P1L1	16	141	6.5	NS	GOOD
255	MUNIYAMMA	28	2485	G3P2L1	20	146	5.1	NS	GOOD
256	LAVANYA	26	1478	G3P2L2	18	148	4.2	NS	GOOD
257	JOTHIKA	29	2589	G3P2L2	20	147	5.8	NS	GOOD
258	SARANYA	30	1747	PRIMI	17	142	5.9	NS	POOR
259	PONKOLALI	24	1569	PRIMI	21	148	5.5	NS	GOOD
260	REKHA	27	2589	G2A1	22	147	5.4	NS	GOOD
261	THILAKAVATHI	20	3256	G3P2L2	16	145	5.2	NS	GOOD
262	PUSHPALATHA	29	1478	G2P1L1	17	149	5.6	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
263	PONMANI	24	1256	G3P2L2	21	147	5.9	NS	GOOD
264	RASAMMA	28	2589	G2P1L1	22	149	5.8	NS	GOOD
265	RAVATHI	27	2458	G2A1	18	147	5.5	NS	GOOD
266	MURUGAMMA	23	1478	G3P1L1	19	145	5.7	NS	GOOD
267	RAKI	30	1569	G2P1L1	21	142	5.9	NS	GOOD
268	JANANI	31	3256	G2p1l1	23	146	5.1	NS	GOOD
269	FATIMA	34	3589	G5p4l3	16	143	5.2	NS	GOOD
270	SHABEENA	28	1247	G2p1l1	19	148	5.5	NS	GOOD
271	SHAN	25	1589	G2a1	18	149	5.4	NS	GOOD
272	REVATHI	29	2698	G2P1L1	20	147	5.3	NS	GOOD
273	REHIMA	26	3147	G2P1L1	22	146	5.9	NS	GOOD
274	MANGALAM	29	1258	G3A2	16	142	5.8	NS	GOOD
275	INDIRA	31	1789	G2P1L1	18	147	5.6	NS	GOOD
276	FARITHA BANU	32	3698	G3A2	20	143	5.8	NS	GOOD
277	RASATHI	28	2563	PRIMI	23	147	5.1	NS	GOOD
278	THASEEN	29	1478	G2P1L1	22	145	5.4	NS	GOOD
279	SANTHA	30	2589	G3P2L2	24	148	5.5	NS	GOOD
280	MARY	24	3258	PRIMI	16	149	5.2	NS	POOR
281	ROHINI	28	1478	G2P1L1	18	142	6	NS	GOOD
282	REKHA	29	2589	G2P1L1	21	146	5.5	NS	GOOD
283	REVATHI	24	3256	PRIMI	16	141	5.5	NS	GOOD
284	JERINA	20	1896	G2P1L1	18	145	5.7	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
285	TEENA	24	2596	G2P1L1	19	148	5.9	NS	GOOD
286	HEMAVATHI	21	1478	PRIMI	17	147	5.8	NS	GOOD
287	MOHAN	25	3259	G2P1L1	20	146	6	NS	GOOD
288	REUNUKADEVI	29	1478	G3P2L2	23	149	5.2	NS	GOOD
289	SOMUNDESHWARI	25	2596	PRIMI	22	147	5.4	NS	GOOD
290	SAGAYAM	28	3698	G2P1L1	24	146	5.2	NS	GOOD
291	VANITHA	30	2365	G3P2L2	21	145	6.5	NS	GOOD
292	GOWRI	29	2147	G3P2L2	17	143	5.1	NS	GOOD
293	BHAVANI	24	3698	PRIMI	19	142	5.6	NS	GOOD
294	SUGANDI	26	2147	G2P1L1	17	149	5.7	NS	GOOD
295	RANJITHA	28	1589	PRIMI	18	148	5.9	NS	GOOD
296	VASANTHI	26	2563	G3A2	16	145	5.5	NS	GOOD
297	ESWARI	21	3698	G2P1L1	21	149	5.6	NS	GOOD
298	RENU	28	1478	G2P1L1	23	147	5.8	NS	GOOD
299	SHALIMMA	24	2569	G2P1L1	21	148	5.7	NS	GOOD
300	DEEPA	27	1426	G3P1L1A1	19	146	5.5	NS	GOOD
301	ELAYARASI	25	2569	PRIMI	17	142	5.9	NS	GOOD
302	RAGARANI	30	2145	G2P1L1	16	143	5.8	NS	POOR
303	JAYANTHI	28	3315	G2P1L1	20	141	5.6	NS	GOOD
304	MALATHI	21	2478	PRIMI	23	149	5.8	NS	GOOD
305	ROHINI	24	1589	PRIMI	22	148	5.6	NS	GOOD
306	RATHA	29	1478	G2P1L1	20	147	5.9	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
307	CHANDRIKA	25	1369	G2P1L1	23	145	5.8	NS	GOOD
308	MUTHULAKSHIMI	29	2658	G3P2L1	16	146	5.8	NS	GOOD
309	THANGAM	24	3698	G2P1L1	19	142	6	NS	GOOD
310	ELAMMA	21	1254	PRIMI	18	143	5.5	NS	GOOD
311	MALATHI	25	1584	G2P1L1	16	147	5.4	NS	GOOD
312	MALAVI	30	1478	PRIMI	19	149	5.2	NS	GOOD
313	SHARMILA	31	2547	PRIMI	18	145	5.1	NS	GOOD
314	SANGAVIROJA	28	2698	G2P1L1	17	146	6	NS	GOOD
315	PONNI	26	3258	G2P1L1	18	143	5.5	NS	GOOD
316	THENMOZHIS	24	1478	PRIMI	20	142	5.6	NS	GOOD
317	KALAIYARASI	32	5478	G3P2L2	23	148	5.9	NS	GOOD
318	VANI	21	6985	PRIMI	21	149	5.8	NS	GOOD
319	KARISHNAVENI	25	2145	G2P1L1	19	145	5.1	NS	GOOD
320	SHANTHI	24	1458	G2P1L1	17	147	5.7	NS	GOOD
321	JASMINE	26	2569	G2P1L1	18	149	4.1	NS	GOOD
322	JAYA	28	2478	G3P2L2	20	145	6	NS	GOOD
323	NASEEMA	21	3258	PRIMI	16	146	5.5	NS	GOOD
324	NAGALAKSHMI	24	3698		18	145	5.6	NS	GOOD
325	MEENA	26	2145	G2P1L1	19	148	5.9	NS	GOOD
326	GAYATHRI	28	2458	G2P1L1	17	149	5.7	NS	GOOD
327	RENUKA	21	1589	PRIMI	20	145	6	NS	GOOD
328	KANIMOZHI	23	1478	PRIMI	23	142	5.6	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
329	RENU	28	1369	G3P2L2	21	143	6	NS	GOOD
330	ROSHMA	24	2145	G2P1L1	23	149	5.5	NS	GOOD
331	ELAVARASI	29	2458	G3P2L2	22	148	5.6	NS	POOR
332	SARMILA	26	1258	G2P1L1	24	**147**	**5.1**	**NS**	GOOD
333	SULOKSANA	24	2365	PRIMI	19	142	6	NS	GOOD
334	MANIMALA	28	2478	G2P1L1	18	143	6	NS	GOOD
335	GOVINDAMMA	27		G2P1L1	16	145	5.5	NS	GOOD
336	NIRMALA	21	1254	G2P1L1	17	149	5.6	NS	GOOD
337	VIJI	30	1589	G3P2L2	19	147	5.9	NS	GOOD
338	PRIYA	31	3697	G2P1L1	20	146	5.6	NS	GOOD
339	SREYA	25	2589	G2P1L1	21	145	6	NS	GOOD
340	STELLA	26	1478	G3A2	18	148	5.3	NS	GOOD
341	POONIMA	28	1369	G2P1L1	19	147	6	NS	GOOD
342	NANTHINI	21	1452	PRIMI	18	145	5.9	NS	GOOD
343	SUSEELA	24	1254	G2P1L1	17	142	5.1	NS	GOOD
344	SUJITHA	27	1256	G2P1L1	20	143	5.9	NS	GOOD
345	JOTHI	25	1232	PRIMI	23	146	5.2	NS	GOOD
346	ANALAKSHMI	29	1265	G3P2L1	21	149	5.5	NS	GOOD
347	MANGAYARKARASI	23	1452	PRIMI	22	148	6	NS	GOOD
348	MANIMAGALAI	24	1452	PRIMI	24	146	5.9	NS	GOOD
349	JENIFER	21	1265	PRIMI	19	149	5.7	NS	GOOD
350	YASMINE	25	1524	G2P1L1	18	147	5.5	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
351	YOGALAKSHMI	32	1265	G3P2L1	17	142	5.8	NS	GOOD
352	MANJULA	21	1542	PRIMI	16	143	5.7	NS	GOOD
353	ANJALI	25	1426	G2P1L1	21	146	5.9	NS	GOOD
354	AROKYAMARY	28	1246	G3P2L2	24	148	6	NS	GOOD
355	AMSAVENI	24	1426	PRIMI	19	147	5.1	NS	GOOD
356	MEEAKSHI	26	1246	G2P1L1	18	149	5.5	NS	GOOD
357	BABY	21	1426	PRIMI	20	142	5.2	NS	GOOD
358	SUDARKODI	24		PRIMI	23	149	5.4	NS	GOOD
359	MANIMEGALAI	23	1452	G2P1L1	21	147	5.8	NS	GOOD
360	TAMIL	21	1524	PRIMI	20	142	5.5	NS	GOOD
361	KOKILA	24	1452	G2P1L1	23	148	6	NS	GOOD
362	MARY	28	1453	G2P1L1	16	149	5.2	NS	GOOD
363	MUTHULAKSHMI	24	1265	G2P1L1	24	142	5.6	NS	GOOD
364	SRIDEVI	26	1365	G3P2L2	18	145	5.4	NS	GOOD
365	TAMARAI SE	28	1364	G3P2L2	20	147	5.6	NS	GOOD
366	RANI	25	1326	PRIMI	19	148	5.9	NS	GOOD
367	MUNIYAMMA	20	1263	PRIMI	16	149	5.8	NS	GOOD
368	RAGAVI	25	1325	G2P1L1	20	142	5.6	NS	GOOD
369	NAMITHA	26	1236	G2P1L1	19	147	5.7	NS	POOR
370	SHAKILA	23	1524	PRIMI	21	146	5.9	NS	GOOD
371	SALOMIYA	25	1265	G2P1L1	23	148	5.8	NS	GOOD
372	BAVANI	21	2145	PRIMI	19	142	5.6	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
373	BHARANI	32	2195	G3P2L2	18	141	5.5	NS	GOOD
374	LALLLU	26	2587	G2P1L1	21	149	5.6	NS	GOOD
375	LAVANYA	28	1256	G2P1L1	22	145	5.4	NS	GOOD
376	MAGESHVARI	25	3256	PRIMI	20	147	6	NS	GOOD
377	KALYANI	29	2155	G2P1L1	21	142	5.3	NS	GOOD
378	VELAMMMAL	25	1478	PRIMI	23	148	5.2	NS	GOOD
379	KANNIGA	24	2145	G2P1L1	21	142	5.9	NS	GOOD
380	VALAR	25	2478	PRIMI	19	141	5.7	NS	GOOD
381	VIJAYA	29	3256	G2P1L1	18	143	5.6	NS	GOOD
382	KOKILA	24	1478	PRIMI	17	149	6	NS	GOOD
383	VENDA	28	1562	G2P1L1	20	147	5.6	NS	GOOD
384	VALARMATHY	24	4569	PRIMI	18	145	5.1	NS	GOOD
385	VIJAYALASHMI	26	2145	PRIMI	17	148	5.8	NS	GOOD
386	SUNDARI	29	2547	G2P1L1	18	142	5.5	NS	GOOD
387	KANMANI	21	1458	PRIMI	20	147	5.2	NS	GOOD
388	GAYATHRI	22	2598	PRIMI	22	146	5.1	NS	GOOD
389	MYTHILI	24	1458	G2P1L1	19	149	6	NS	GOOD
390	DHANAM	25	2369	G2P1L1	18	148	5.5	NS	GOOD
391	SIVAGAMI	28	2154	G3P2L2	23	145	5.9	NS	GOOD
392	KANCHANA	29	1478	G2P1L1	24	142	5.7	NS	GOOD
393	REVATHI	24	1458	PRIMI	18	149	5.6	NS	GOOD
394	SANGAVI	25	1478	G2P1L1	17	147	5.9	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
395	RENU	26	1895	G2P1L1	20	145	5.7	NS	GOOD
396	NISH	21	1245	PRIMI	16	149	5.8	NS	GOOD
397	BABY	25	2458	G2P1L1	20	147	6	NS	GOOD
398	MUMTAJ	21		PRIMI	18	142	5.5	NS	GOOD
399	KANIYAMMA	24	1458	G2P1L1	19	146	5.8	NS	GOOD
400	JAYANTHI	26	1425	G2P1L1	21	141	5.9	NS	GOOD
401	MALAR	24	2548	PRIMI	23	145	6	NS	GOOD
402	KUMARI	32	2654	G3P2L2	18	149	5.3	NS	GOOD
403	GANTHIMATHI	30	2148	G2P1L1	17	148	5.5	NS	GOOD
404	MALATHI	25	2158	PRIMI	22	142	5.8	NS	GOOD
405	SELVI	26	1547	G2P1L1	20	146	5.9	NS	GOOD
406	SHANTHI	28	1598	G2P1L1	19	141	5.8	NS	GOOD
407	NOORBI	29	1289	G3P2L2	17	148	6.1	NS	GOOD
408	MASTHAN	24		PRIMI	23	143	5.3	NS	GOOD
409	PADMAVATHI	25	1547	PRIMI	21	149	5.5	NS	GOOD
410	GIRIJA	22	1358	PRIMI	20	145	5.9	NS	GOOD
411	POLAMMAL	21	1298	PRIMI	19	147	6	NS	GOOD
412	LAKSHMI	25	3014	G2P1L1	13	145	4.8	NS	GOOD
413	AMBIKA	24	3088	PRIMI	20	148	5.2	NS	GOOD
414	GRACE	22	3725	PRIMI	21	143	6	NS.	GOOD
415	SHEEBA	28	3645	G3A2	24	148	5.6	NS	GOOD
416	LALITHA	32	3212	G2P1L0	23	149	5.2	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
417	LILLY	29	3423	PRIMI	26	145	5.7	NS	GOOD
418	JEBA	21	7625	G2A1	24	142	5.3	NS	GOOD
419	RAMYA	24	3545	PRIMI	23	146	5.4	NS	GOOD
420	FATHIMA	23	3654	G2P1L0	24	149	5.7	NS	POOR
421	PRIYA	24	3767	G3P2L1	26	145	5.8	NS	GOOD
422	DEVI	27	3645	PRIMI	22	143	5.4	NS	GOOD
423	ANNAM	21	3256	G2P1L1	26	148	5.6	NS	GOOD
424	VEMBU	25	3897	G2A1	25	143	5.7	NS	GOOD
425	KANIKA	21	3745	PRIMI	24	150	5.2	NS	GOOD
426	VANI	32	3264	PRIMI	25	151	5.7	NS	GOOD
427	NITHIYA	36	2546	G2P1L1	24	143	5.7	NS	GOOD
428	RAJI	26	3612	PRIMI	26	145	5.9	NS	GOOD
429	DIVIYA	19	3765	G2A1	24	142	5.3	NS	GOOD
430	PUSHPA	23	3243	G3P2L2	23	147	5.2	NS	GOOD
431	USHA	27	3906	PRIMI	26	148	5.7	NS	GOOD
432	MARIAM	24	3712	G2P1L1	25	147	5.3	NS	GOOD
433	ANITHA	27	3456	G2P1L1	24	146	5.6	NS	GOOD
434	AMBIKA	21	2434	PRIMI	26	143	5.2	NS	GOOD
435	SATHIYA	21	3762	PRIMI	21	142	5.3	NS	GOOD
436	VANI	27	3154	G3P1L1A1	23	145	5.4	NS	GOOD
437	MANJU	33	3651	G2A1	27	150	5.8	NS	GOOD
438	RAJI	22	3871	G2A1	24	143	5.4	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
439	KUMARI	19	3908	PRIMI	26	147	5.6	NS	GOOD
440	NATHIYA	23	3710	PRIMI	26	149	5.6	NS	GOOD
441	KOWSEI	26	3004	G2P1L1	24	148	5.3	NS	GOOD
442	KAIALB	21	3612	G4A3	23	146	5.2	NS	GOOD
443	BHARTHI	24	3817	G2A1	25	143	5.1	NS	GOOD
444	THARANI	32	3945	G3P1L1A1	25	150	5.4	NS	GOOD
445	KGUMARI	24	3732	PRIMI	24	146	5.5	NS	GOOD
446	ANITHA	21	3767	PRIMI	23	145	5.2	NS	GOOD
447	SUJI	26	3434	G3A2	24	145	5.8	NS	GOOD
448	VALI	19	3624	G2P1L1	24	143	5.5	NS	GOOD
449	REMYA	28	3898	G2A1	24	147	5.1	NS	GOOD
450	SUBBAMMAL	23	1452	PRIMI	22	148	6	NS	GOOD
451	JOYCE	24	1452	PRIMI	24	146	5.9	NS	GOOD
452	RENGANAYAGI	21	1265	PRIMI	19	149	5.7	NS	GOOD
453	SHYNI	28	3909	G2P1L0	25	148	5.3	NS	GOOD
454	BLESSY	19	3709	G2P1L1	26	145	5.2	NS	GOOD
455	JAMILA	22	3203	G2A1	23	146	5.3	NS	GOOD
456	MALIKA	27	3150	PRIMI	21	146	5.4	NS	GOOD
457	SHANTHI	32	3265	PRIMI	27	146	5.8	NS	GOOD
458	JAYA	20	4512	G2P1L1	25	143	5.4	NS	GOOD
459	MARY	30	2369	PRIMI	21	142	5.5	NS	GOOD
460	MALI	19	5125	PRIMI	23	147	5.3	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
461	RAJI	22	2356	G2A1	25	146	5.6	NS	GOOD
462	SHEELA	28	2969	G2P1L1	24	149	5.6	NS	GOOD
463	RAGA	24	1569	G3P1L1A1	26	143	5.6	NS	GOOD
464	AJIM	26	2365	PRIMI	27	148	5.7	NS	GOOD
465	JAYA	27	4865	PRIMI	23	142	5.4	NS	GOOD
466	ELAVARASI	25	2314	PRIMI	25	147	5.5	NS	GOOD
467	LENI MARY	22	1856	G2A1	24	145	5.9	NS	GOOD
468	JANCY	26	5369	G2P1L1	24	145	5.8	NS	GOOD
469	ALMELU	28	4236	G3P2L2	26	146	5.6	NS	GOOD
470	SHALINI	29	2658	G2P1L1	25	145	5.3	NS	GOOD
471	RATHNA	26	2159	G2P1L1	25	147	5.2	NS	GOOD
472	SIVRANJANI	32	3698	PRIMI	26	147	5.5	NS	GOOD
473	JANCYRANI	34	2489	PRIMI	23	148	5.6	NS	GOOD
474	KALIVANI	22	2178	PRIMI	23	145	5.8	NS	GOOD
475	NANTHINI	26	2695	G2A1	24	143	5.3	NS	GOOD
476	KANMANI	29	3125	G3P1L1A1	25	145	5.1	NS	GOOD
477	SAROJA	25	3254	PRIMI	25	142	5.3	NS	GOOD
478	GAYATHRI	32	2985	G2P1L1	24	143	5.2	NS	GOOD
479	KAVITHA	24	2569	G2P1L1	26	149	5.4	NS	GOOD
480	KANIKA	29	2489	G3A2	23	147	5.4	NS	GOOD
481	CLARA	27	2658	G2A1	27	147	5.4	NS	GOOD
482	SHEELA	21	2478	PRIMI	24	145	5.3	NS	GOOD

S.No	NAME	AGE	IPNO	OBSTETRIC CODE	GESTATIONAL AGE(WEEKS)	GCT mg/dl	HbA1C (%)	USG	OUTCOME
483	THARA	23	2698	PRIMI	25	146	5.6	NS	GOOD
484	LEELAVATHI	21	3112	PRIMI	23	145	5.7	NS	GOOD
485	THANGAMMA	24	3276	G2P1L1	16	141	5.5	NS	GOOD
486	SOBANA	32	2145	PRIMI	24	145	5.7	NS	GOOD
487	KAVIYA	34	2458	G2A1	27	145	5.7	NS	GOOD
488	KIRTHEEKA	26	2698	PRIMI	25	143	5.4	NS	GOOD
489	JAYANTHI	28	2478	G2P1L1	24	148	5.3	NS	GOOD
490	SOLOKCHANA	26	3248	G2A1	24	148	5.2	NS	GOOD
491	SUGANTHI	21	3125	G3P1L1	21	149	5.2	NS	GOOD
492	MALARGODI	25	4865	G3P2L2	23	145	5.9	NS	GOOD
493	RAVATHI	26	2985	PRIMI	26	147	5.2	NS	GOOD
494	SUGANYA	23	2487	PRIMI	25	146	5.3	NS	GOOD
495	RATHIKA	24	2698	G2A1	24	143	5.4	NS	GOOD
496	SANGEETHA	21	2147	G2A1	24	148	5.4	NS	GOOD
497	CHANDRA	29	2985	G2P1L0	23	148	5.9	NS	GOOD
498	MANGALA	26	3248	PRIMI	24	148	5.6	NS	GOOD
499	SELVI	28	2890	PRIMI	22	147	5.4	NS	GOOD
500	GEETHA	25	3345	PRIMI	24	143	5.6	NS	GOOD